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VOL. CLI, No. 4 APRIL, 1916 No. 529

THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES

GEORGE MORRIS PIERSOL, M.D., EDITOR
JOHN H. MUSSER, JR., M.D., ASSISTANT EDITOR

JUN 3 1944

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Philadelphia LEA & FEBIGER New York

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THE

AMERICAN JOURNAL

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APRIL, 1916.

ORIGINAL ARTICLES

CONGENITAL AND ACQUIRED ENURESIS FROM SPINAL LESION. (a) MYELODYSPLASIA. (b) STRETCHING OF THE CAUDA EQUINA.

By William G. Spiller, M.D.,

Professor of Neurology in the University of Pennsylvania.

Myelodysplasia. Under the name of myelodysplasia, Alfred Fuchs,[1] in 1909, described anomalies of development, and enuresis nocturna, associated often with spina bifida occulta, and depending on imperfect development of the lower part of the cord. The important features of this condition are:

1. Weakness of the sphincters and especially enuresis nocturna persisting after puberty.

2. Syndactylism between the second and third toes, more rarely between the second, third, and fourth toes, still more rarely between the other toes; usually bilateral.

3. Disturbances of sensation, chiefly of temperature sensation, not strictly radicular in type, especially in the feet and more frequently only in the toes.

4. Defect of the sacral canal recognized by the Roentgen-rays.

5. Anomalies of cutaneous and tendon reflexes in the abdomen and lower limbs.

6. Defects in the feet in many cases (pes planus, varus, valgus), sometimes with peroneus weakness, also trophic and vasomotor disturbances in the toes.

[1] Wien. med. Wchnschr., 1909, p. 2141.

Other anomalies that should be sought for are hypertrichosis of the sacral region, lipoma in the coccygeal region, asymmetry of the rima ani, fovea coccygea, or fistula-like depression of the sacrococcygeal region.

Mattauschek studied with Fuchs 24 adults with enuresis (20 soldiers and 4 civilians) and found anomalies in 21, which seemed to indicate hypoplasia of the conus.

Peritz and Saar observed by means of the Roentgen rays in 20 adults with enuresis and 20 children afflicted with the same disorder that 68 per cent. of the adults and 35 per cent. of the children had spina bifida occulta.

Lewandowsky has objected that spina bifida and enuresis are merely associated conditions (cited by Vorkastner.)[2]

Peritz[3] believes that these conditions occurring in the same person have a relation to one another, because anomalies of the sacrum are found in 68.2 per cent. of cases of enuresis in adults; because of thermo-anesthesia on the plantar surface of the toes, and of anomalies in the reflexes with enuresis. He states that 50 to 55 per cent. of children who have enuresis nocturna have it because of myelodysplasia.

Alfred Saenger,[4] since the report made by Fuchs, has examined every case of enuresis nocturna with roentgenographs, but has found only one in which myelodysplasia existed. His patient was a girl, aged eighteen years, who had complained of nocturnal enuresis a long time. She had no difficulty in holding the urine during the day. The enuresis had increased in later years. The right Achilles reflex was absent, a zone of diminished sensation in all forms was about the anus, and the Roentgen-ray picture revealed a defect of the sacrum.

Most cases of enuresis nocturna, he thinks, can not be attributed to myelodysplasia.

An unsigned (except for the initials J. C.) review of the myelodysplasia of Fuchs has been published recently in the *Archiv. de méd. des enfants*, August, 1915. Reference is made to three observations recently reported by C. Bonorino-Udaondo and Mariano R. Castex.[5] As these cases were observed in Buenos Aires and are published in an inaccessible journal I have abstracted them from the review.

Case I.—Boy, aged fourteen years, had nocturnal and diurnal incontinence of urine since early infancy. The diurnal incontinence disappeared about the age of six or seven years, but the nocturnal incontinence persisted, and occurred only in sleep. He had certain malformations, cephalic and facial hypertrichosis, ogival palate,

[2] Handbuch der Neurologie, Lewandowsky.
[3] Deutsch. med. Wchnschr., July 6, 1911. p. 1256.
[4] Deutsch. Ztschr. f. Nervenh., xlvii and xlviii, 694.
[5] Review de la Soc. méd. Argentina, November and December, 1914.

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skeletal malformations, syndactylism of the feet, sensory disturbances confined to the big toes, and congenital valvular disease of the mitral orifice with hypertrophy of the right ventricle.

Motility was well preserved in the four limbs and trunk. The tendon and cutaneous reflexes were normal. Sensation was normal, except that thermic sensation was considerably diminished in the dorsal and plantar surfaces of the big toes. An interdigital membrane between the second and third and the third and fourth toes of each foot was pronounced (syndactylism).

Palpation over the vertebral column in the lumbar region revealed at the lower part of this region a slight oval depression, measuring 3 cm. in vertical diameter and 2 cm. in transverse diameter. Pressure here caused severe pain, and gave the examiner the impression of fibrous and not bony tissue. The defect implicated the lower lumbar vertebræ and sacrum, and was well shown by the Roentgen-rays. The pelvis was also deformed. The Wassermann reaction of the blood was positive.

CASE II.—Youth, aged twenty years, had a condition similar to that in Case I. He had nocturnal enuresis, small forehead, ogival palate, general and sexual infantilism, syndactylism, disturbance of sensation in each big toe, and congenital mitral valve lesion.

CASE III.—Boy, aged eleven years. He had pain in the legs, nocturnal enuresis from time to time, infantilism, thermic anesthesia of the big toes, syndactylism in each foot, and defect of the lowest lumbar vertebra and sacrum.

In all three cases the findings were: nocturnal enuresis, skeletal malformations, infantilism, ogival palate, cephalofacial hypertrichosis, syndactylism, and sensory disturbances in the big toes.

Whether defective development of the lower parts of the cord without defect in the sacrum and lumbar vertebræ is a common cause of enuresis nocturna with or without the described anomalies of development can not be determined without necropsies, and as yet sufficient attention has not been directed to this subject to enable us to form positive conclusions.

It is important to remember that the enuresis of older children and adults, not necessarily merely nocturnal, may occasionally be a sign of spina bifida occulta, and may exist without other signs or with few signs of this defect. Examination of the sacrum and lower lumbar vertebræ by means of the Roentgen-rays will readily determine a defect in these parts if it exist, and such a defect should warrant the physician in assuming that much exercise of the lower limbs might be followed by muscular palsy in the peroneal nerve supply. This is true in cases where pronounced spina bifida occurs, as in a case referred to me by Dr. Edward Martin, March 27, 1909. Had the young man avoided training for severe rowing he might not have developed all the serious symptoms he presented. The evident spina bifida should in itself have been a warning.

Still more important is the second case, in which the statement made by the boy that for years he had had enuresis led me to request a Roentgen-ray photograph of the sacrum and lower lumbar vertebræ from Dr. Pancoast. This patient gave no appearance of spina bifida to the sight or touch of the examiner, except some tenderness on deep pressure over the sacrum, but the Roentgen-rays showed a serious defect of the sacrum and lower lumbar vertebræ. The paralysis that developed from the comparatively light exercise of a bicycle ride of an hour and a half might have been avoided had the spina bifida occulta been suspected from the persistent enuresis.

CASE I.—*Pronounced spina bifida with enuresis and other symptoms increased by violent exercise.*

A. B., aged eighteen years. About two years previously to 1909 he had been in training in rowing, and he thought he had overtrained. It had become necessary for him, probably because of the training, to urinate often, and he had difficulty in holding the urine. He was obliged to urinate once every hour or even oftener. About 1905 he noticed that the toes of the left foot were becoming flexed, and the tendons of the toes and the Achilles tendon were cut. The left lower limb became weaker than the right and the left leg below the knee smaller. The right lower limb had not been affected. The bowels were regular, the power of erection was not affected, and seminal emissions at night occurred about once every three weeks. He had never had sexual intercourse, and there was apparently no implication of the nerve supply for the sexual functions.

Dr. Martin had obtained the statement that the urine was discharged in gushes resembling the rhythmical contractions of the gluteal muscles. A short time before my examination the patient on one occasion had tonic spasm of the abdominal muscles, at which time he had difficulty in getting his breath.

I found the left lower limb below the knee, including the foot, much smaller than the right, and there were marked fibrillary tremors below the left knee on the inner side of the leg. These were not observed elsewhere. The left toes were partially flexed. The left calf measured 30.5 cm., the right calf 35.5 cm. in their greatest circumference. The left thigh was a little atrophied in its inner part. Slight diminution of touch, pain, and temperature sensations was observed over the right buttock near the anus, but the impairment of these sensations was greater over the left buttock and in a band down the back of the left thigh as far as the knee. The patellar reflex was a little diminished on each side, and the Achilles reflexes were lost. There was no Babinski reflex. The limbs showed no abnormal tonicity, and there had not been pain. A spina bifida was observed over the sacrum. When the young man consulted me he was having constant dribbling of urine.

CASE II.—*Spina bifida occulta with enuresis and other symptoms developing after moderate exercise.*

R. C., aged fourteen years, was referred to the University Hospital by Dr. F. Bloomhardt, of Altoona. He was at first in Dr. Stengel's service, but was kindly referred to my service.

About May 1, 1915, the boy noticed some weakness in both feet. He could not run well, the toes dragged a little on the ground, but no illness preceded the onset of these symptoms. His family, however, noticed no disturbance until Memorial Day, at which time, after a three-mile bicycle ride, the boy found that the weakness of the feet was becoming pronounced, but he had no weakness at the knees or hips. The condition gradually improved slightly, and he recovered some motion in the toes and ankles, especially in the right leg. He never had any pain or paresthesia. When he came to the hospital he stated that he had always had difficulty in retaining the urine, although he never had pronounced incontinence. He had incontinence of feces on one occasion.

The examination about four months after the bicycle ride showed that all the symptoms aside from the enuresis were confined to the lower limbs. The muscles of the thighs were in fair condition. The legs below the knees were somewhat wasted and there was bilateral foot-drop. The power of spreading the toes had been lost. Dorsal flexion was almost abolished in both feet, and plantar flexion was impaired in the left foot. The patellar tendon reflexes were diminished and the Achilles reflexes were lost. Tactile sensation was normal. Pain sensation was impaired along the outer edge of the sole of the left foot and on the plantar surface of the toes.

The back in the lumbar and sacral regions appeared to be entirely normal to sight and palpation, except that slight tenderness could be elicited by deep pressure over the sacrum. The Roentgen rays showed a grave defect of the sacrum and lower lumbar vertebræ.

In this connection reference may be made to an interesting case of spina bifida reported by Tutyschkin, Neurologisches Centralblatt, February 1 and 15, 1914.

ENURESIS FROM STRETCHING THE CAUDA EQUINA. Disturbance of micturition may be acquired by stretching of the lower sacral roots in bending the trunk far forward on the lower limbs. In such a position the lower roots are severely stretched, and although the bending of the spinal column may be chiefly in the midthoracic region, the lower roots of the spinal cord are likely to be more affected than any others. These roots are the longest and the stretching is more severe in them. An interesting example of this accident is shown by the following case:

CASE III.—J. C., aged forty-six years, was referred to the University Hospital October 12, 1915, by Dr. J. R. Thompson, of Pittston. About nine or ten years ago while working in the shaft of a mine and stooping over, an elevator descended upon him and

he was caught beneath it. He sustained very severe pressure in such a manner that his trunk was bent forward on the lower limbs and his sight was affected for a time, but he was not made unconscious. He was taken home and kept on a lounge for several days, but was not paralyzed in the lower limbs. He returned to the mine about a week after the accident, walking with the aid of a stick.

Difficulty in retaining the urine and feces began soon after the accident and persisted about a year. He had incontinence of urine at night at times, but now has no difficulty in retaining the urine unless he has taken cold. He gets up once or twice at night to urinate, and during the day he voids urine more frequently. He has no oss of sexual desire, but power of erection has been lost, and he has no seminal emissions. The Achilles reflexes are lost, the right patellar reflex also is impaired, and the left patellar reflex seems a little exaggerated. He has some indication of Romberg's sign. Pain and tactile sensations about the anus and in the perineum are normal, as in the lower limbs.

A case that I reported in 1907[6] before the Philadelphia Neurological Society is similar to the one just described. The view has been held by some that hemorrhage might be the cause of the symptoms in such a case, but it is difficult to understand why the hemorrhage should occur only in the conus of the cord when the arching of the body is above this region, and in a case where the symptoms are unilateral, as in the following, hemorrhage seems improbable. Of interest in this connection is the report by Bittorf[7] of a man who while striking a heavy blow on an object between his feet missed the object and the blow went farther than he intended and caused paralysis of the sciatic nerve, probably from stretching of the nerve in the bending of the body forward, although hemorrhage into the nerve may have occurred.

H. C., a male, aged thirty-one years, was injured eighteen months previous to my examination by a bale of cotton falling against the abdomen. He was unable to work for about three weeks, but then returned to heavy work, feeling not quite so well as formerly. About a month after returning to his occupation, while lifting a bale of cotton, he "felt something give way" in the right inguinal region, and at the same time he heard a tearing sound. It is uncertain what this was. He immediately felt weak and limped on the right lower limb, but walked home, a distance of about two blocks, and went to bed. After one day he got out of bed but remained at home about a week. He then returned to heavy work, but was not so strong as he had been before the injury.

After the accident he lost control of the bladder, so that when he

[6] Jour. Nerv. and Ment. Dis., 1907, p. 665.
[7] Semaine méd., December 26, 1906, p. 616.

coughed or exerted himself the urine would escape. This condition became worse gradually until he had no control of the bladder, and was obliged to wear a urinal. Sexual desire was not weakened, but the dribbling of the urine prevented the sexual act. The rectal sphincter functionated feebly and a call to stool was urgent. His gait and station were good. The lower limbs were well developed, but the man felt weaker than before the accident. The left side of the scrotum, the left side of the perineum, and the left buttock near the anus had fully normal sensation to touch and pin prick; whereas the right side of the scrotum except the upper outer portion, the right buttock in a small area near the anus, and to a less degree the right side of the perineum, showed diminished sensation to touch and pin prick. The right side of the penis also was less sensitive than the left side. The sensation of the testicles was normal. The patellar reflexes were prompt but the Achilles reflexes were slight. Babinski's sign was not present. The upper part of the body was not affected. The lesion seemed to be in the lower sacral roots, and may have been confined to one side because of the unilaterality of the disturbance of sensation in the supply of these roots. This unilaterality seemed to indicate that the lesion was not in the conus. The cause of the symptoms was probably stretching of the lower sacral roots of one side by excessive straining in lifting a heavy weight while bending forward.

GELATINOID CARCINOMA (MORBUS GELATINOSUS) OF THE PERITONEUM.[1]

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The special interest in this case is concerned with several points: (1) It is an example of ascites existing over a long period and requiring a large number of tappings. (2) The problem of diagnosis. (3) The character of the peritoneal fluid. (4) The interpretation of the autopsy findings.

The history is as follows: G. B., a colored man, aged thirty-eight years, a butcher by occupation, was admitted to the Jefferson Hospital first on February 21, 1911.

[1] Read at the meeting of the Association of American Physicians, May, 1915.

Family History. His father died at fifty-eight years from asthma, his mother at sixty years, the cause not being known; he has six brothers and sisters living and well; one died from an accident and one from typhoid fever. There is no history of tuberculosis.

Personal History. Measles, mumps, and chicken-pox in childhood were the only infectious diseases. There is no history of cough or shortness of breath. The appetite and digestion have been good. There was no history of any illness suggestive of appendicitis. There is no note of any urinary trouble. He had a gonococcus infection at the age of eighteen, but gave no history of a luetic infection. The patient had used only small amounts of alcohol.

Present Illness. In the summer of 1910 the patient noticed there was some enlargement of the abdomen. This occurred without any pain or disturbance of the stomach or bowels. The distention did not last long and the patient states that it disappeared entirely. In January, 1911, the swelling of the abdomen returned and increased gradually until he was admitted to the hospital. There had been no pain, and the patient complained only of discomfort when coughing or stooping over. He had some shortness of breath on severe exertion, but worked until five days before admission. Otherwise he had been in good condition and did not complain of any gastric or intestinal trouble.

Examination. The patient was well nourished. There was no definite jaundice. The lungs showed dulness at both bases and the heart was displaced upward. These were both regarded as being due to the pressure from the abdomen. The abdomen showed very marked enlargement and the usual signs of ascites. The liver and spleen could not be felt. There was no edema of the scrotum and very slight edema of the legs. The blood count showed hemoglobin, 68 per cent.; reds, 4,900,000; leukocytes, 7400. The urine had a specific gravity which varied from 1.015 to 1.030, and generally contained a small amount of albumin, but was otherwise negative. The abdomen was tapped and about nine liters of fluid removed. This presented the usual appearance of ascitic fluid, and was not peculiar in any way. The specific gravity was 1.030, and there was a considerable amount of albumin. The majority of the contained cells were lymphocytes. After the tapping the fluid reaccumulated for a time and then diminished. He was discharged with evidence of some fluid in the abdomen, but in good general condition. The picture at this time was apparently that of cirrhosis of the liver with ascites.

During the following three years the patient was admitted to the hospital seventeen different times. It does not seem necessary to mention these in detail, and only the important points are given, which, for convenience, may be put under separate headings.

General Condition. The patient's general health remained extremely good. The only complaint which he made on each return to the hospital was of the abdominal distention. It was only on his last admission, March 4, 1914, that any change was noted. He had then lost weight and was suffering from frequent vomiting, which apparently prevented his getting sufficient nourishment.

Abdominal Condition. This was much the same on each admission. The degree of distention varied somewhat, but was always quite marked. The usual physical signs of ascites were always present. There were no signs suggesting any disease of the gall-bladder, appendix, or cecum. Examination of the rectum did not show any abnormality.

Abdominal Tapping. Up to the time of his last admission he had been tapped fifty-eight times. On forty-six occasions fluid was obtained, the amount of which varied from 2500 c.c. to 10,560 c.c. During a period of about three years from the onset, he was tapped nine times and the fluid had always the same character as noted on the first occasion. On the tenth tapping (May 22, 1913) a different state of affairs was found, which will be referred to later. One curious point was that on several occasions he was much better after an attempted tapping which was not successful. On two admissions there was no successful tapping, but the patient was discharged very much improved and with the distention lessened.

The patient, naturally, became an authority on the methods of being tapped, and always graded the different house officers under whose care he came, according to their ability to tap. He emphasized two characteristics which the trocar should have, namely, that it should be sharp and of large size.

On May 22, 1913, there was a distinct change in the material obtained by tapping. On this occasion a number of gelatinous masses escaped with the ascitic fluid. This was about three years after the onset and about one year before death. These gelatinous bodies varied in size from about the size of a grain of wheat to an ordinary pea; occasionally larger masses were obtained. Their appearance was gelatinous and in some ways rather suggested boiled tapioca grains. The larger masses that were obtained had much the same appearance. Occasionally the trocar was completely blocked with these, but, as a rule, they did not interfere materially with the tapping. About a month later, as tapping was again required, it was thought well to do an exploration, and a small incision was made in the abdominal wall. A large quantity of fluid was obtained and the same gelatinous colloid-like material. The opening was not large enough to permit of any extensive investigation of the abdomen, but nothing abnormal in any of this viscera was noted through the small incision. The fluid at this

time showed 83 per cent. of small lymphocytes, 11 per cent. cells with large nuclei, and the remainder polynuclear cells and others difficult to classify.

The problem of diagnosis was a most interesting one. The patient had chronic ascites, but his general condition was excellent and there was no jaundice. In August, 1913, the abdomen was markedly distended, with definite evidence of ascites, but this was not extreme. For the first time enlargement of the liver was found and the edge of the left lobe was felt in the midline 3 cm. above the navel. It seemed quite distinctly irregular. It was not possible to feel the edge of the right lobe with certainty. The spleen was considerably enlarged and readily felt below the costal margin. In view of the long duration of the ascites, the fact that it had come and gone, the patient's excellent general condition, and the enlargement of the left lobe of the liver and spleen, the question of syphilis of the liver had to be considered. However, the Wassermann reaction was always negative and no signs of syphilis were to be found unless the abdominal condition was due to it. Still it seemed worth while to let the patient have some potassium iodide, and it was begun at this time. We never felt sure that it made any difference, although the patient claimed that after taking it the fluid accumulated much more slowly. During this admission the patient was tapped a number of times, the fluid always containing the same gelatinous masses.

Examination of the cells in the ascitic fluid showed considerable variation in the percentage of lymphocytes at various tappings, and also in the occurrence of eosinophiles. At this time (August, 1913) it was not possible to be certain that the right lobe of the liver was enlarged, but a little later this was definitely made out, and in January, 1914, the edge in the right nipple line was felt 10 cm. below the costal margin. The enlargement of the left lobe previously noted was still present. So far as we could determine there had been a definite increase in the size of the right lobe. In view of the fact that he had been taking potassium iodide quite steadily for some months, this was strongly against the possibility of the enlargement of the liver being due to lues. The last admission was on March 4, 1914. By this time his general condition showed marked change, shortness of breath and edema were more marked, and he had been much troubled by vomiting. Examination of the stomach contents did not show anything of special interest except that no free hydrochloric was obtained. The blood showed only a moderate secondary anemia; the differential count was normal. The obstinate constipation which he had on admission was soon replaced by persistent diarrhea. We tested the length of time which it took for material to pass through the bowel and found that this was usually about four or five hours. When the condition of the intestines was seen at autopsy this seemed very remarkable.

The difficulty of tapping increased so that sometimes it was necessary to tap in several places before we could obtain any fluid. There were eleven successful tappings on the last admission. At times he complained of severe abdominal pain, but did not show any marked tenderness. It was not possible to feel either the liver or the spleen at this time. About two weeks before death a number of *nodular masses* were felt in the abdomen. These were evidently in the peritoneal cavity, felt quite superficial, were very hard, and quite freely movable. They varied in size from a diameter of about half an inch to perhaps one and a half inches. They were felt in different places on different days. The vomiting and diarrhea continued until death on May 7, 1914.

The main facts may be summarized as follows: A male, aged forty-one years, with chronic ascites for four years and fifty-seven successful tappings. For three years the fluid showed no peculiar features and then gelatinous colloid-like material appeared and was present for a year until death. During the greater part of this time the patient's general condition was excellent. The liver and spleen showed enlargement at one time and later became reduced in size. Shortly before death many hard, rounded, movable masses were felt in the abdomen. There was intractable diarrhea for the last few weeks of life.

The gelatinoid material was studied in the laboratory of our colleague, Professor Hawk. It was a mucin-like substance, much like the material known as serosa mucin. It contained 11.5 per cent. of nitrogen and 0.8 per cent. of sulphur.

The question arises as to the course of events, and it must be confessed that the autopsy did not throw much light on this. For three years the patient had ascites regarded as due to cirrhosis of the liver. But cirrhosis of the liver without some other condition does not, as a rule, permit of repeated tappings over such a long period, for ascites is usually a terminal event in pure cirrhosis. With the appearance of the gelatinoid material in the ascitic fluid the question arose whether the process had been of a malignant character from the onset. If so it must have been extremely mild. The presence of ascitic fluid with the characteristics of that found in cirrhosis of the liver has been noted for some time before the appearance of signs suggesting a malignant process. Thus in the case reported by Pye-Smith, during a period of ten months the patient was tapped seven times and the usual fluid of ascites secondary to cirrhosis was found. On the next tapping cells were found which suggested colloid carcinoma. This was the celebrated case in which tapping was done 299 times. Death occurred after a duration of nearly nine years, and at autopsy a papillomatous tumor of each ovary was found. The peritoneum was thickened and covered with secondary papillomata.

Another obscure point was that three months after the first

finding of gelatinoid material the liver and spleen were found to be enlarged and six months later the enlargement was still more marked. Subsequently the liver decreased in size, and before death the liver and spleen could not be felt. There does not seem to be any explanation for the increase in the size of the liver and spleen. The enlargement was not secondary to circulatory failure. The size of the liver at autopsy was proof that we had not been mistaken in the belief that the enlargement had disappeared. In the absence of cirrhosis it seems probable that the decrease in size of the liver was due to the marked increase in the thickness of the capsule. It may be suggested that the enlargement did not actually exist but the liver was very easily felt after tapping and the increase in size occurred under observation. The position of the upper limit of dulness did not change.

PATHOLOGICAL REPORT.[2] The body is that of a poorly nourished adult colored male. *Rigor mortis* is present but easily broken up. Pupils equal and contracted. Slight edema of both ankles. Abdomen greatly distended and tense. Subcutaneous fat is very scanty. Abdominal and thoracic musculature is pale red and greatly atrophied.

As matters of special interest pertain to the abdomen a brief survey only will be given to the other findings:

Left pleura slightly thickened; cavity contains a few cubic centimeters of slightly turbid, yellowish fluid. Between the lobes of the left lung, situated immediately under the pleura, are several elveated grayish areas, 5 to 10 mm. in diameter, which on section show a lobular arrangement, the alveoli containing a grayish transparent gelatinoid substance. Although the lung appears small it crepitates throughout except a narrow stratum at the base; the lower lobe is also somewhat congested.

Right pleura is dry; the serosa slightly thickened, especially over the base. On the pleural surface of the diaphragm are a few grayish gelatinous bosses and granulations; the largest of the former measures a little over 1 cm. in diameter; the fine granulations may be exudative or neoplastic, of which one cannot be sure. The nodules in the muscular portion of the diaphragm have raised the serosa, which is still, apparently, intact. Except for a few areas of partial collapse and considerable congestion, a large part of which is clearly hypostatic and associated with some edema, the lung is not the seat of any noteworthy abnormality. In neither lung was any secondary growth observed. No metastasis in the mediastinal lymph nodes or other tissue except the subpleural growths already mentioned.

Pericardium uninvolved. Heart is displaced upward and slightly

[2] Section and preliminary examination made by Dr. E. D. Funk, demonstrator of morbid anatomy, upon whose protocol the following is in part based. Much of the general description deemed immaterial to this presentation is omitted.

to left. No important lesion. There are a few negligible sclerotic areas in aorta and larger branches.

Except for a mild patchy fibrosis the kidneys are normal. No gross change in adrenals. No noteworthy lesion in bladder or genitalia.

Permission to examine the central nervous system was not obtained.

Abdomen and Contents. As the incision approaches the abdominal cavity it is noted that the parietal peritoneum is thickened, at points almost leathery, and in such areas resists incision. When the abdomen is widely opened the almost universal presence of a gelatinoid material is observed. It has accumulated particularly in the pelvis, lumbar regions, right and left, and between the stomach and liver. About 2500 c.c. were removed; all that would adhere to visceral and parietal peritoneum was left in position; it was thought that formalin fixation would secure it in place, but to a slight degree only was this result attained. The greatly thickened parietal peritoneum was dissected loose from the muscles on each side and posteriorly, the diaphragm incised along its attachment to the parietes, and the entire mass removed for fixation and further study. The following data were obtained from examination of the fresh specimen, and later, after formalin fixation, by such dissection as was permissible without destroying the specimen (see Fig. 1).

The upper border of the specimen is formed by the diaphragm, which on the inferior surface is intimately attached to every viscus with which it comes in contact. The attachments to the liver and spleen are particularly firm. The intestines, including the colon, and the stomach are closely packed in the median line and matted together by adhesions, many of which are so firm that the enclosed viscus tears before the adhesion yields. Nothing that could be certainly identified as omentum remains distinguishable in the mass. Between the stomach and liver is a cavity holding more than a liter of gelatinoid substance. This has displaced the stomach downward and rather behind the colon to which it is firmly attached. The transverse colon is greatly dilated, the dilatations being most marked near the hepatic and splenic flexures. To this part of the colon is also attached the small intestine, heaped in an indistinguishable mass. With a long brain knife the anterior portion was cut away, exposing the opened convolutions of the intestines beneath. With difficulty these were separated above, as shown in the illustration, to permit palpation of the pancreas, which does not appear changed. The caput coli is greatly dilated and the wall slightly thickened. The outer aspect of the caput coli and a part of the descending colon and sigmoid are attached to the parietal peritoneum. The ascending colon passes mesially almost to the midline behind the small intestine and ascends tortuously to the hepatic flexure. A similar mesial displacement of the descending

colon is also present. Apparently these displacements are due to pressure of gelatinous masses occupying the lateral aspect and

FIG. 1.—Contents of abdomen. Anterior aspect. Carcinoma of peritoneum. Upper margin formed by liver and attached diaphragm. Just below liver, between that organ and stomach, cavity containing gelatinoid material; cavities to right and left and also pelvis contained similar material. Every part of serosa in contact with any other surface is adherent. Some of these adhesions have been broken up. Intestines are rigid and stand open on section. Note nodules on abdominal wall, especially to lower right. For further description see text.

lumbar region of the abdomen. The area of the gall-bladder and pylorus is occupied by a dense fibroid mass containing many small cavities filled with gelatinoid material.

Incision through 5 cm. of this mass opens the gall-bladder; the cavity is 9 cm. long and irregularly compressed with a maximum breadth of 3 cm. It is filled with bile-stained gelatinoid material somewhat thinner than much of that in the peritoneum, but may be scooped out as a semisolid mass, dark greenish-yellow in color. The mucosa is smooth. The bile ducts were not dissected.

The liver is small, capsule greatly thickened; adhesions practically universal to all surfaces. Several incisions disclosed no secondary growth. Microscopically the cells are granular and shrunken, with a slight increase in fibrous tissue, most notable beneath the capsule.

The appendix was found buried in a mass of adhesions, and though not fully dissected is about the thickness of the little finger and admits a probe 8 cm. The connection with the cecum is closed and the base of the appendix bulges into the cecum as an ovoidal boss 2.2 cm. in diameter. On incision it is found to contain the same gelatinoid material as that present in the abdominal cavity and gall-bladder. The obvious thickening is around the appendix rather than of the appendix; the musculature and mucosa are not thicker than normal, if anything, at least in areas, they appear thin. The great and irregular thickening of the parietal peritoneum is well shown in the illustration. Just below the right lobe of the liver the thickening extends through the peritoneum and into the aponeurosis of the muscle. Neither macroscopically nor microscopically is the wasted muscle found involved. The inner surface of the parietal peritoneum is covered with loosely attached gelatinoid or colloid material; relatively little of this substance is shown in the illustration. There are also, at many points, on the parietal peritoneum and on the adhesions binding the viscera, numerous nodules varying in size from 2 to 3 mm. to 4.5 cm. In the illustration two of these are well shown at the lower right on the parietal peritoneum, and on the mass of adherent intestines in the median line near the lower border. Masses of exactly the same type are also free in the peritoneal cavity; several of these were removed, attached on a frame, and photographed (Fig. 2). Incised in the fresh condition they were found to possess outer capsules 1 to 3 mm. in thickness, and to contain soft gelatinoid material which flowed from the cut surfaces with about the same consistency as thick egg albumen. Fixed in formalin they retained their contour and bore incision without loss of contents. Several of those shown in Fig. 2 exhibit the essential morphology. Running in from the capsule are numerous fine delicate trabeculæ without regularity of arrangement, forming a sort of a reticulum, in the meshes of which is contained the gelatinoid substance. Numerous histological sections show these to be composed of imperfectly developed fibrous tissue containing a few round cells, an occasional leukocyte, and homogeneous staining gelatinoid material. In

none of the sections was any epithelium encountered. It was evidently these structures that could be palpated through the abdominal wall during life. Several were still loosely attached

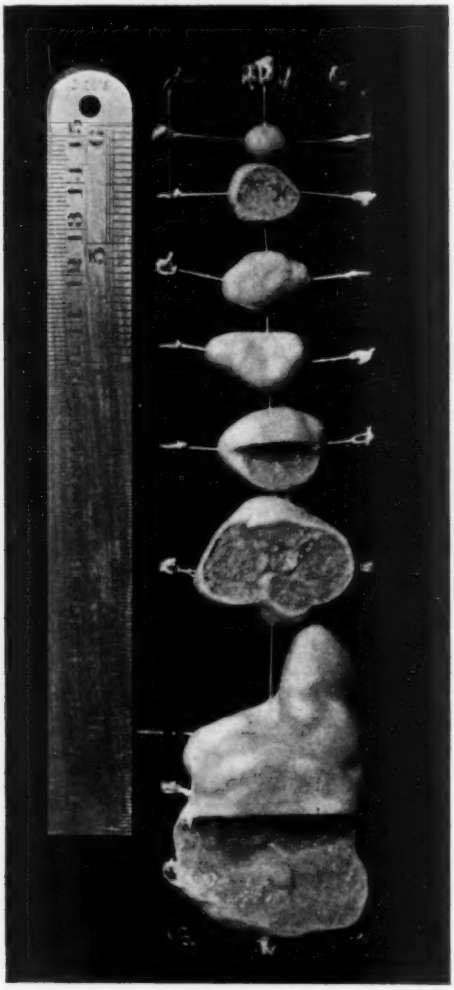

FIG. 2.—Nodules from peritoneum. Many of these, of practically every size, the largest 5 cm. in diameter, were free in the belly cavity or loosely attached, often by a thin filament only, to the abdominal wall. Those shown were all free. For further description see text.

and apparently about to separate from the wall or the visceral mass. Examination of one of the attached masses seemed to indicate that they arise from condensation of the gelatinoid material

FIG. 3.—Section of parietes. Abdominal wall immediately below liver mass. Photomicrograph. *A*, muscle of abdominal wall; *B* to *C*, muscle fascia, extraperitoneal tissue, and peritoneum in single thick layer of dense fibrous tissue, at points separated by interstitial edema; *C* to *D*, stratum in which neoplastic growth is at points demonstrable. Just above leader from *C*, stratum of columnar epithelial cells, most of which are of the tall pallisade type. Note that just beneath epithelium edema is marked; *D*, and above, reticulum partly enclosing spaces containing homogeneous gelatinoid material; *E*, space clearly outside peritoneum; a few disappearing epithelial cells along upper margin, others below; space filled with gelatinoid substance; square outlined in centre is shown under higher magnification in Fig. 4.

which later becomes edematous, the characteristic cellular elements disappearing. Their development is entirely internal to the structure that was thought to occupy the position of the normal peritoneum. Ssobolew observed similar bodies.

Morbid Histology. Sections of the organs are for the most part without interest. Several of the gelatinoid masses removed at operations and others obtained postmortem, were fixed and stained by appropriate methods without disclosing any cellular element upon which a histological diagnosis could be based. Similar disappointing results were obtained from numerous examinations made of parts removed from several areas in the parietal peritoneum and from the thickened masses of adhesions. Most of these showed an exceedingly edematous fibrous reticulum obviously in various stages of formation. The general character of the histological appearance may be gathered from a close study of the photomicrograph shown in Fig. 3, which includes the edge of the abdominal muscle below; above is the denser fascia, the peritoneum showing the neoplastic involvement, and at the top the reticulum surrounding irregular communicating spaces containing the homogeneous, almost cell-free, gelatinoid substance. It will be observed that the latter contains a scanty cellular detritus and nothing upon which a histological diagnosis as to the character of the process could be based. However, in the examination of a large number of sections numerous pictures similar to that shown in the centre of the field were encountered. Note the papillary growth projecting upward surmounted by tall columnar epithelium the cell nuclei of which are marginated immediately at the bases of the cells. The higher magnification of this same area is shown in Figure 4. Many of the epithelial cells are of the chalice type. The connective tissue immediately underlying the cells is, in practically every instance, obviously edematous, and apparently when the edema reaches a certain stage the cell disintegration begins and continues until the epithelium entirely disappears.

The morphology and chemical reactions of these cells are identical with those observed in the palisade epithelium commonly found lining the varieties of ovarian cysts with which this disease is so frequently associated; no one who has examined the structure has any doubt as to the epithelial origin. Identical appearances in growths obviously connected with the appendix have been reported.

In the many sections examined structures possessing the same general characters as those described have been found only occasionally. Such growths, however, have been observed in the extraperitoneal fascia and in the diaphragm immediately beneath both peritoneum and pleura.

The almost universal involvement of every structure in the abdominal cavity in this case, and the absence of any conclusive dominance in one particular area renders impossible an unassailable statement as to the origin of the process. Those who believe strongly in its appendicular relation will lay stress on the fact that the appendix is involved. An examination of Fig. 1 must

impress the observer with the fact that the gall-bladder area is conspicuously affected. At this point the colon, gall-bladder, intestine, pyloric complement of stomach, diaphragm, and abdominal parietes are implicated. If finding gelatinoid material in the cavity of a viscus could be taken as proof of origin in that organ, there would be some ground for the statement that in our patient the process might here have arisen in the gall-bladder. Had the pri-

FIG. 4.—Small central area from Fig. 3 more highly magnified. Photomicrograph. In centre papillary growth covered with tall columnar epithelium; many cells are of the chalice type. Part of another papillary growth is shown at left. To right note regressing epithelium; edema at base of papillary mass and beneath palisade epithelium.

mary lesion been in the appendix one would not have expected so extensive an involvement of the upper abdomen. Of course, without more detailed section than has been made it has been impossible to exclude the stomach, duodenum or pancreas, or indeed for that matter almost any part of the intra-abdominal alimentary canal. It is quite certain that such a tumor might have originated in a diverticulum. In the absence of later and more exact information concerning the nature of these growths the condition would

probably have been regarded as a primary neoplasm of the peritoneum; this possibility, we think, may be excluded.

Of special interest is a consideration of the nature of the process concerning which many views have been held. Pean, forty-five years ago, applied the term gelatinous disease of the peritoneum, fully recognizing and admitting the vagueness of the term. In

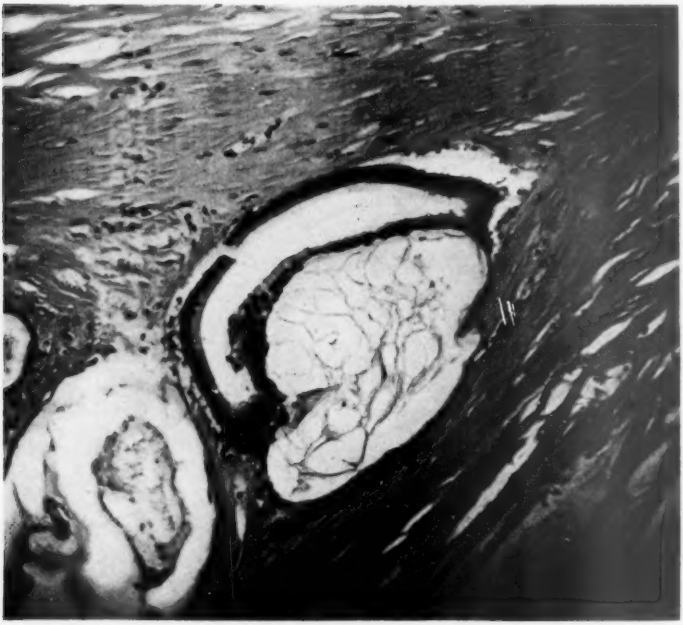

FIG. 5.—Section from fibrogelatinoid area in gall-bladder region. Photomicrograph. Typical epithelial lined space showing regression of epithelium and edematous gelatinoid substance arising beneath the epithelial cells; over this substance the epithelium is disappearing. Below and to left (left lower corner) is a mass of gelatinoid substance containing two regressing epithelial cells. It has not been possible with certainty to determine the origin of this gelatinoid substance. Just below centre of left margin part of vessel in stratum of fibrous tissue. A few fibroblasts and an occasional leukocyte may be recognized.

some form no doubt this was what Virchow called colloid cancer and to which has also been applied the terms mucoid cancer, gelatinous ascites, and carcinomatous ascites. Werth, in 1884, introduced the name pseudomyxoma, to which there are many objections, but which unfortunately has been widely adopted, and under which name most of the cases are recorded. We believe the process is neoplastic and essentially cancerous, but would manifest no par-

ticular preference between the terms colloid, mucoid, muciparous, or gelatinoid carcinoma, if anything, inclining toward the last. The designation "maladie gelatineuse" is not inappropriate. Failure to recognize the carcinomatous nature of the process rests upon the fact that endless search or exceptional good fortune in any particular case may be necessary to locate the areas of definitely cancerous morphology. The fact that invasion extends through the peritoneum into the muscle fascia, through the diaphragm from serosa to serosa, with the formation of subpleural nodules, the nodules between the lobes of the lung, and other evidences seem to us difficult of explanation except on the assumption that the process is potentially malignant.

The universality of the retrograde changes in the epithelium, not commonly encountered in cancer, is due to some influence which our study has failed fully to disclose. Apparently the proliferating epithelium is encountering some antagonistic influence that is constantly breaking it down. The epitheliolytic agent seems to be of humoral origin. No evidence of phagocytosis, no noteworthy leukocytic infiltration, no giant cells, are present, but an edema, in which the epithelium seems to undergo disintegration and solution without leaving behind the usual detritus incident to chromatin fragmentation commonly observed in the presence of a necrosis and cell disintegration. In Fig. 5 it can be noted that beneath the epithelium on the under side of the space lined by cylindric cells there is a collection of edematous gelatinoid material over which the epithelial cells are manifesting retrograde changes. In a space below and to the left the epithelium has almost completely disappeared, and this retrogressive change on the part of the epithelial cells is manifest in almost every location where they are found. The influence that brings it about seems to come after the cell is growing or in some instances after the cell is fully grown, suggesting that the growth of the cell is necessary for its appearance. Following conventional usage it would be easy to suggest a name for such a hypothetical body; at the risk of being prosaic we forego this.

It is well established in this class of cases that complete removal of early manifestations may be curative, and that admittedly incomplete excision of advanced lesions may be followed by long intervals before recurrence takes place and in some instances apparent recovery. Such cases have been recorded by Lejars, Trotter, Neumann, Eden and others. In one of Lejars's cases the tumor disappeared. In Eden's patient gelatinous disease due to an ovarian tumor was present at operation done "several months" after appearance of symptoms; the ovary and a large part but not all of the jelly were removed; three months later the patient was well. Two years and four months after the first operation the other ovary and appendix were found involved and removed.

Nearly two years later the patient was reported well. According to Lejars, lesions of ovarian origin are more malignant than those arising in the appendix.

Destructibility of cells composing or derived from malignant tumors has been established by numerous observers, and more recently has been especially investigated by Iwasaki. Twenty years ago Goldmann suspected the presence of some protecting body. Hodenpyl's results obtained by injecting ascitic fluid derived from a patient with cancer aroused hopes that have not materialized. Our case and many in the literature more than suggest the existence of some such body and of tissue and humoral potentialities that we may in the future find some way to mobilize. The peritoneum may be able to produce such an agent. Bland-Sutton says "large numbers of cancer cells which are shed into the abdominal cavity perish, for the peritoneum exercises defending or prophylactic power against such marauders." We know that tissues engaged in processes essentially protective in nature suffer, often are almost destroyed when the fight is lost; note the lymph-nodal changes in many infections, such as plague and tuberculosis, the liver necroses in infections and intoxications and consider the destroyed omentum in some of these peritoneal neoplasms. Possibly that organ of inconclusively-established function may be playing a part of which we know nothing. According to Theilhaber there are on record 200 cases of cancer spontaneously cured. Coley's results in sarcoma cannot be ignored. The problem is to determine by what mechanism the result is attained.

BIBLIOGRAPHY.

In the preparation of this article the following have been consulted:

Apolant. Jour. Exp. Med., 1911, xiv, 316.

Bland-Sutton. Brit. Med. Jour., 1912, i, 1170.

Boidin. Man. de mal. du tube digestif, Debove, Achard, and Castaigne, Paris, 1908, ii, 563.

Bristowe. Diseases of the Intestine and Peritoneum, New York, 1879, p. 220.

Chavannaz. Bull. et mém. d. l. Soc. de chir., Seance April 21, 1909, p. 459.

Coley. Compt. rend. du 24° Congrès de l'Assoc. Française de Chir., 1911.

Courtois-Suffit. Traite d. méd. Bouchard and Brissaud, 1900, iv, 608.

Cramer. Monatssch. f. Geburtsh. u. Gynäk., December, 1910.

Cramer. Semaine méd., 1911, p. 187.

Delétrez. Bull. de l'Acad. royale de méd. d. Belgique, iv series, 1913, tome xxvi, p. 341.

Duval. Ann. de méd., January, 1914.

Eden. Lancet, 1912, ii, p. 1498.

Elbe. Beitr. z. klin. Chir., 1909, lxiv, p. 635.

Fleisher, Vera, and Loeb. Jour. Exp. Med., 1914, xx, 522.

Fraenkel. Münch. med. Wchnschr., 1901, p. 965, and 1912, lix, p. 1142.

Frank. Proc. New York Path. Soc., 1911, n. s., xi, 2.

Hartman and Kindley. Jour. Am. Med. Assn., 1914, lxii, 1795.

Hodenpyl. Med. Record, February 26, 1910.

Hueter. Ziegler's Beitr., xli, 1907.

Iwasaki. Jour. Path. and Bact., 1915, vol. xx.

Lahey and Haythorn. Amer. Jour. Med. Sci., February, 1912.

Lejars. Semaine méd., 1912, xxxii, 589.
Levin. Jour. Exp. Med., 1911, xiv, 139; No. 4, xiii, 604.
Levin and Sittenfield. Jour. Exp. Med., 1911, No. 4, xiii, 511; xiv, 148.
Lunckenbein. München. med. Wchnschr., September 2, 1913.
MacCarthy and McGrath. Surg. Gyn. and Obstet., March, 1911.
McConnell. International Clinics, vol. ii, 20th series.
Merkel. Lubarsch and Ostertag's Ergebnisse, 1903, Abt. 2, S. 965.
Merkel. Müchnen. med. Wchnschr., 1904, p. 1360.
Müller. Inaug. Dissert., Griefswald, 1913.
Oberndorfer. Verhandl. d. deutsch. path. Gesellsch., 1906, p. 235.
Ogilvie. Jour. Am. Med. Assn., 1915, lxiv, 657.
Paczek. Wien. klin. Runsch., July 6, 1913.
Pean, cited by Goursolas. Thèse de Lyon, 1911.
Pye-Smith. Trans. Path. Soc., London, 1893, xliv, 116.
Rous and Murphy. Jour. Exp. Med., 1912, xv, 270.
Russell. Fifth Scientific Report on the Investigations of the Imperial Cancer Research Fund, London, 1912.
Sabrazès. Compt. rend. Soc. de biol., Paris, 1911, ii, 474.
Ssobolew. Frankft. Zitschr. f. Path., 1913, xiii, 344.
Theilhaber. Deutsch. med. Wchnschr., June 27, 1912.
Trotter. Brit. Med. Jour., 1910, i, 687.
Tsurumi. Jour. Path. and Bact., 1915, vol. xx.
Turner. Brit. Med. Jour., 1912, i, 229.
Weil. Jour. Exp. Med., 1913, xviii, 390.
Weil. Jour. Med. Research, August, 1910, p. 85.
Werth. Arch. f. Gynäk, 1884, vol. xxiv.
Wilson. Lancet, 1912, ii, p. 1496.
Notes chirurgicales: Semaine méd., 1912, No. 31, p. 368.

THE CLINICAL RELATIONS OF GRAVITY, POSTURE AND CIRCULATION.

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The vascular mechanism is everywhere in a state of "tone" by which its capacity is actively reduced. Were the physiological tone to be released the capacity of the blood channels in the splanchnic area alone would be sufficient to contain all the blood and the circulation would come to an end in the erect posture because no fluid could reach the heart.

This tonicity of the vascular bed is what keeps the blood from stagnating in the capacious reservoirs of the abdomen under the influence of gravitation. It is essentially a vital phenomenon. There is a distinction between the regulating mechanisms for the preservation of tone in the arteries as contrasted with the veins of the splanchnic system, but an essential adjunct to the proper function of the veins is the mechanical support afforded by the normal tension of the abdominal wall.

Physiologists have supplied us with fundamental data for the apprehension of the hydrostatic disorders of the circulation.

The experiments of Leonard Hill[1] are especially rich in significance. Hill secured animals (dog, cat, monkey) upon a board which could be rotated in a vertical plane. The animal could then be readily turned from the horizontal to either a feet-down or feet-up posture. Both arterial and venous blood-pressures were graphically recorded throughout the experiments.

It was found in the four-footed animal that the carotid blood-pressure always fell in the feet-down posture, and rose in the feet-up posture, as compared with the horizontal. The fall of arterial pressure in the first case was due to the accumulation of blood in the splanchnic veins and a correspondingly lessened inflow into the heart; the higher arterial pressure in the last case depended on a plethora resulting from excessive emptying of the abdominal veins.

In a small dog changing from the feet-down to the feet-up posture caused the carotid pressure to vary from 16 mm. Hg. below to the same amount above the normal. In certain of the animals, especially in the monkey, the hydrostatic effects of changed posture were compensated and the carotid pressure remained normal; in the feet-down posture there was active splanchnic vasoconstriction and in the feet-up posture the reverse. Chloroform anesthesia tended to disable the mechanism of compensation. Under chloroform narcosis the splanchnic vasomotor mechanism was paralyzed and in the feet-down posture the blood from the general circulation would gradually collect and stagnate in the splanchnic veins, the heart become empty and ceased to beat. If in such a case it was attempted to revive a heart by resorting to the head-down posture the inrush of blood would overdistend and paralyze the weakened organ. Chloroform prevents the normal contractile reaction of the heart. A bandage tied around the abdomen or pressure applied with the hands readily compensated for the fall of blood-pressure in the feet-down posture by preventing the accumulation of blood in the veins. Sudden removal of pressure upon the abdomen in the feet-down position could drain the heart and kill the animal.

In general, according to Hill, any influence which weakens the splanchnic vasomotor mechanism or, the writer would add, that lowers the contractile tone of the abdominal wall, magnifies proportionately the postural effects of gravity on the circulation.

These postural changes in the carotid blood-pressure are of profound importance in determining the blood-pressure and circulation within the brain. On account of the rigidity of the skull the brain remains, according to Hill, practically at a constant volume. Its vessels are never, while the cranium is intact, drained of blood, but the circulation may stagnate in the brain of feet-down animals. When in such a posture a trephine hole is made in the

[1] Jour. Physiol., 1895, xviii, 15.

skull the brain at once collapses, becomes drained of blood, and the animal quickly dies. Hill had an opportunity to verify on a man with a trephine hole in his skull the intracranial pressure changes dependent on posture. He calls attention to the danger in elevating the head of a patient with a defect in the skull when the vasomotor mechanism is incompetent.

Bayliss, Hill, and Gullard[2] declare that "the brain has no direct vasomotor mechanism, but its blood supply can be controlled indirectly by the vasomotor centre acting on the splanchnic area." They find that "the intracranial pressure in all physiological conditions remains the same as the cerebral venous pressure. The intracranial pressure is wholly circulatory in origin and may vary with the circulatory from zero to 50 to 60 mm. Hg. The functions of the brain matter continue with the varying conditions of pressure." Under "physiological conditions a rise of arterial pressure accelerates the flow of blood through the brain and a fall slackens it. . . . The rise of arterial pressure that occurs in pathological conditions of increased intracranial pressure is not protective but tends to increase the extent of the cerebral anemia. The right direction of treatment therefore in such conditions is to lower the blood-pressure." Though strong evidence is furnished by other experimenters that the vessels of the brain are not devoid of vasomotor fibers the main contentions of the authors quoted hold good.

Hill and Barnard[3] dwell especially on the application of these data in the human subject. "In a man six feet high the hydrostatic pressure of a column of blood reaching from the vertex to the sole of the feet equals about 140 mm. Hg., and from the vertex to the middle of the abdomen about 50 mm. Hg. . . . The splanchnic vasomotor mechanism is by itself amply sufficient to compensate for the hydrostatic effect of gravity. . . . In man a condition of deficient vasomotor tone combined with an atonic condition of the abdominal wall or patulous abdomen must in the erect posture lead to a deficient circulation and anemia of the brain."

Surgical shock furnishes the most startling exhibition of the vital disaster attending circulation failure in the intact vascular circuit. Passing the extensive literature dealing with the pathology of this condition the writer will assume as proved that view according to which acute traumatic shock has its origin neither in heart failure nor vasomotor failure. This conclusion in no way surrenders the notion that vasomotor failure may have an important etiological bearing on the conditions with which this paper deals.

Basing his views on animal experimentation, Yandell Henderson[4] announces the theory that shock, both surgical and toxemic, depends upon the accumulation of blood within the large veins of

[2] Jour. Physiol., 1895, xviii, 334.
[3] Ibid., 1897, xxi, 323.
[4] Amer. Jour. Physiol., 1910, xxvii, 152.

the abdomen, from which reservoir it fails to flow into the heart. The animal behaves as if bled to death within his own vessels. According to Henderson the large abdominal veins are normally maintained in a state of tone not by vasomotor nerves, but through an intrinsic venopressor mechanism whose efficiency essentially depends upon the tension of CO_2 and of the acid-alkali balance in the fluids of the body. This conception of the chemical and osmotic regulation of the venopressor mechanism of the splanchnic area suggests that any departure from healthy metabolism may cause virtual dilatation of the abdominal reservoir and lead to stagnation of blood within it when the erect posture is assumed.

From the purely clinical point of view, T. C. Janeway[5] some years ago clearly pointed out that those familiar catastrophes which we were wont to designate as "heart failure" are, for the most part, not heart failures at all but vascular failures put into effect by the postural drainage of the blood into the "abdominal reservoir." While in the normal man or animal the bloodless heart might easily be revived by restoring the vital current, in the toxemic, shocked, or anesthetized subject it could easily happen that a brief period of bloodlessness would fatally reduce cardiac irritability.

Hill[6] has shown that efforts to restore the circulation under shock conditions by the feet-up posture are prone to result in paralysis of the heart by overdistention. Clinicians are familiar with the hazards involved to patients with debilitated hearts when the bowels are moved in the sitting posture; especially, probably, is this the case during states of flux. The special danger lies, presumably, in a diminished splanchnic tone on these occasions.

A most important corollary to all these teachings is the expression of the unity of the circulatory system. There is no rational study of heart disease by itself. The efficiency of the heart depends as intimately upon the efficiency of the bloodvessels, their nerves, and of tissue metabolism and osmosis as upon the integrity of its own structures, and all must be taken into account by the philosophic physician.

The mechanical conditions surrounding the circulation in the brain and in the abdomen are in significant contrast. In the former the rigidity of the skull keeps the organ relatively full of blood even with a zero of arterial pressure. Under such a condition there must be exerted upon the brain tissues molecular tensions which may have profound physiological importance, but of whose mode of action we are wholly ignorant. In the abdomen, on the other hand, the bloodvessels find less support, perhaps, than in any other situation. The physiological integrity of the splanchnic vasomotor system and of the musculature of the abdominal wall form the chief or only extraneous limitations to expansion of the

[5] New York Med. Jour., 1907, lxxxv, 193.

[6] Loc. cit.

great "abdominal reservoir." Numberless facts support the conclusion that the vascular bed of the abdomen is the great variable in the irrigating system of the body, now contracting, now expanding, to meet the needs of the tissue soil. Simple as might seem the relations of the physical factors which determine pressure within the abdomen, nowhere is more obvious the futility of the deductive method in physiological investigation. Emerson[7] summarized the literature on the nature and conditions of intra-abdominal pressure up to 1911, and was not able to establish any concensus of opinion from the authorities. By some the intra-abdominal pressure is held to be positive, above that of the atmosphere, by others negative, below it.

It is rather generally admitted that the pressure is positive or varies with respiratory movement, increasing with inspiration and decreasing with expiration, and that it becomes negative in subjects having pendulous abdominal walls. Emerson himself, on the basis of animal experiments, concludes that the normal pressure within the abdomen is slightly positive, due to the coördination of numerous physiological factors. The pressure slightly rises or falls with descent or ascent of the diaphragm. Excessive diminution of pressure induced by loss of tone in the abdominal wall, or, on the other hand, untoward increase of pressure, as in meteorism, each reduces more or less critically the efficiency of respiration and circulation.

Burton-Opitz[8] demonstrated experimentally the profound alterations in the extra-abdominal circulation attending comparatively slight changes in intra-abdominal pressure. The tendency of positive pressure, as might have been expected, was to force blood out of the abdominal veins and impede the inflow into them. The resultant effect was to increase the amount of blood circulating through the head and diminish it in the lower extremities.

An extensive and practical study of the abdominal circulation in its clinical relations has been made by Oliver.[9] This author points out again that though the hydrostatic effects of postural change are much more manifest on the veins than on the arteries, still arterial pressures are largely subject to gravitation. In the standing position the blood-pressure in the arteries of the head may be but one-half that in the feet, and the latter pressure may fall one-half in the horizontal posture. He has no hesitation in saying that in the normal subject the arterial pressure in the erect position, sitting or standing, is higher than in the recumbent. That is, splanchnic vasomotor tone normally overcompensates the hydrostatic effect of gravity. Fatigue, disease, laxness of the

[7] Arch. Int. Med., 1911, vii, 754.

[8] Amer. Jour. Physiol., 1914, xxxvi, 66; Arch. f. d. ges. Physiol., 1908, cxxi, 156; ibid., 1908, cxxiv, 469.

[9] Blood and Blood-pressure, 1901.

abdominal wall, or any influence which lowers the vasomotor tone or reduces the external support of the abdominal vessels tends to relatively raise the arterial pressure in the recumbent posture. The reason for this seems obviously to consist in the gravitation of blood to the abdominal veins in the erect and corresponding delivery of it to the general circulation in the horizontal posture.

Oliver states that "overloading of the splanchnic veins, from diminution or loss of tone in the arterioles which feed them, is a common, if not an invariable, fact in all forms of debility. . . . In splanchnic states the normal postural variation of the radial caliber is reversed."

Oliver's method of estimating the amount of splanchnic venous stasis is simple and effective. With the subject in the horizontal position the arterial pressure is measured in an arm, then a maximum weight of twenty-eight pounds, a bag of shot, is equably distributed over the abdomen. Excess of blood in the abdominal veins is thus forced toward the heart and the arterial pressure rises proportionately. Observations made before breakfast, when the vascular tone is high, show little or no rise of arterial pressure with abdominal compression. But for thirty minutes after a meal a weight applied to the abdomen may send the arterial pressure up 17 or 20 mm. Hg. Observations repeated at intervals afterward show less and less elevation of pressure with compression. In the healthy subject at rest, abdominal compression might raise the arterial pressure 18 mm. Hg. After vigorous but not exhausting exercise compression of the abdomen had no effect on arterial pressure, owing to excessive dilatation of the systemic vascular bed. But when exercise was continued to fatigue, loss of splanchnic vasomotor tone was evidenced by rise of arterial pressure when weight was applied to the abdomen.

It is important to understand definitely the nature of the interdependence between the tensions of the abdominal wall and the splanchnic veins. If, as Emerson[10] and others have concluded, the intra-abdominal pressure becomes constantly negative when the abdominal wall is patulous, we must recognize therein a steady force tending to aspirate the blood into the thin-walled veins of the cavity. The commonly associated condition of visceroptosis probably acts in the erect posture to still further lower the pressure when the weight of the organs directly stretches the containing wall. In brief, positive intra-abdominal pressure tends to empty and negative pressure to fill the abdominal veins.

The general conception of the pathological significance of intra-abdominal pressures has been the theme of much fugitive medical literature within the past decade or more. The one idea at the root

[10] Loc. cit.

of most of this writing has been derived from palpation of the abdominal wall. Ptosis of the abdominal organs thus disclosed has been held responsible for a long category of pathological symptoms, and the manufacture of devices for correction of the defect employs a host of artisans from whom we can hardly demand an appreciation of etiology. It is not intended here to make a sweeping denial of the pathological importance of visceroptosis, but to point out that, in the majority of cases, clinicians in attributing symptoms to postural dislocation of the abdominal organs have probably mistaken the shadow for the substance.

The foregoing imperfect sketch of the literature dealing with the postural relations of the abdominal circulation almost reduces to platitude the deduction that loss of tone by the splanchnic vasomotor system and the abdominal wall must result, in the erect posture, in anemia of the brain and oligæmia of the general circulation.

The various debilities which may initiate such a failure of tone are, in turn, intensified by its effects, and the vicious circle thus established constantly contracts at the expense of normal function.

The muscles of the abdominal wall are said to be innervated from the respiratory centre. Much remains to be discovered concerning their coördination with the vasomotor system and their relations to the acid-alkali balance of the blood. It is open to suspicion that the teleological idea behind the abdominal rigidity attending peritoneal inflammation is not more to limit painful friction between surfaces than to reduce blood flow within them. The comforting effect of the ice-bag may depend upon the same result.

Dr. C. D. Mosher[11] describes the great relief experienced by her patients with congestive dysmenorrhea when they lay with hips elevated and pressed firmly over the uterus and breathed deeply. This sort of manipulation is well adapted to relieve pelvic congestion.

My own impression of the clinical importance of postural abdominal stasis was not formulated in advance, but was built up by induction from the observations carried on through many years. The facts under consideration are familiar to every clinician; their relation to symptomatology and the indicated *rationale* of treatment seem so obvious that special exposition might appear superfluous, yet somehow it is clear that medical practitioners in great part either fail to understand the data involved in this situation or misapprehend their significance.

In the normal subject the fall of carotid blood-pressure, which tends to occur on changing from the recumbent to the erect posture, leads to stimulation of the splanchnic vasomotor centre; at the same time the tension of the muscles of the abdominal wall is apparently

[11] Jour. Am. Med. Assn., 1914, lxii, 1297.

increased, as is evidenced by the difficulty found in making a satisfactory palpatory examination of the abdomen with the patient upright. In these ways the hydrostatic pressure tending to surcharge the splanchnic veins may be even overcompensated, so that, with the venous outflow from the brain accelerated by gravity and the arterial inflow kept high by elevation of systemic arterial pressure, the nourishment of the brain may be expected to flourish best in the erect posture.[12]

The important aid given in the standing posture to the flow of blood through the lower extremities by the tension and contraction of great masses of skeletal muscles need not be here considered. Suffice it to say, we seem justified in concluding that when the recumbent posture is assumed, at once all the nervous and muscular activities which were engaged in hydrostatic compensation come to rest and gravitation no longer impedes the return of the blood from the abdomen to the general circulation.

When we turn from the healthy to the debilitated individual we must expect to witness the preponderance of gravity effects proportional to the weakening of the physiological powers which had held it in compensation. Now, in the erect posture, the blood actually stagnates to a degree in the splanchnic veins at the expense of the systemic circulation and the blood-pressure in the brachial artery falls. Changing to the recumbent posture the surplus fluid overflows into the heart and the brachial blood-pressure rises. Simple as the problem may appear, investigators differ in their statements of the facts as to the influence of posture on the arterial blood-pressure in the normal human subject. Oliver[13] states emphatically that in the normal subject the pressure in the radial artery is higher in the erect than in the recumbent posture; he finds the arterial pressure in the sitting posture to average about 10 mm. Hg. more than in the supine. Stephens,[14] on the contrary, in a study of 22 cases, presumably healthy young men, found the systolic blood-pressure invariably higher in the recumbent position. His average postural pressures in mm. Hg. were: standing, 130.8; sitting, 131.7; supine, 150.4. The photographs of his experimental arrangements indicate that the arm in which the pressure was measured was raised considerably above the heart in the erect and supported below it in the supine position.

Erlanger and Hooker,[15] in a careful study of two young men, found that on changing from the recumbent to the erect posture there might be either a slight rise or fall of maximal pressure in the brachial artery, but the characteristic effect of the change was a diminution in the pulse pressure in the erect posture, caused by a rise in the minimal blood-pressure. "If on standing the minimum

[12] *Vide infra* Erlanger and Hooker.
[13] Loc. cit.
[14] Jour. Am. Med. Assn., 1904, xliii, 955.
[15] Johns Hopkins Hosp. Rep., 1904, xii, 145.

pressure rises but little the maximum will fall more; but if the minimum pressure is decidedly increased the maximum may rise." On changing from the recumbent to the standing posture they found a rise of minimal (and mean) pressure, as a rule; a diminution of pulse-pressure, an increase of pulse rate. The reverse changes occurred when, after standing, the recumbent posture is assumed. The pressures found in the sitting posture approached those of the recumbent much nearer than those of the standing posture. When the external temperature was elevated they found the minimal pressure to be lower and the pulse-pressure to diminish less on standing.

They say, "The circulation in the brain is, if anything, improved in the sitting and especially in the standing postures." As will be seen, it is exactly the pathological reverse of this cerebral circulation efficiency to which I attribute the symptoms which suggested this investigation. The complexity of the subject has been sufficiently demonstrated to warn us against deductive reasoning which must be based on premises some of which are unknown. Nevertheless, it is proper to consider the directions in which functional efficiency may be expected to become impaired under definite anatomical inadequacies.

Physiologists have shown that experimental reduction of the blood flow to the brain excites the vasomotor centres, especially of the splanchnic areas, with the obvious effort to increase the intracranial blood flow. We can hardly doubt that in a person whose blood gravitates abnormally into the splanchnic veins there is produced a virtual anemia of the brain capable of setting up a functional disability which may be manifested in a wide variety of ways. Patients belonging to the affected class often complain of great muscular weakness. They are apt to develop the general functional incapacity or perversion which we are prone to group under the head of "neurasthenia." Headache and dizziness in the erect posture are characteristic symptoms. It is as if the nervous centres protested against an inefficient effort, as in eye-strain.

Perhaps the most dramatic case among my records is that of an active business man, about fifty years of age, who had gradually developed annoying headache and dizziness for which the neurologist and aurist failed to effectually account. The symptoms manifested themselves only in the erect posture, disappearing when the subject lay down. The patient presented a dazed appearance. His heart was dilated and the abdomen somewhat protuberant. His blood-pressures were: sitting, 104–62; recumbent, 109–69. There was no obvious arteriosclerosis. The patient was made to stand up and hold a pillow over his abdomen. Standing just behind him with my arms around his body, strong pressure was brought to bear on the pillow over the lower abdomen. At once he declared

himself free from headache and dizziness, which symptoms immediately returned on relaxing the pressure. The experiment was repeated several times with the same result. An abdominal belt was prescribed and was worn during the daytime for about six weeks, with complete relief of symptoms. It is interesting to note that for several months after removal of the belt the patient remained normal. Headache and dizziness then occasionally returned, to be relieved by application of the belt, which was resorted to rather unwillingly because of the local discomfort it gave. Great reduction in the cardiac dilatation accompanied the general improvement.

In many of the cases which seem to belong to this category I had no opportunity to make definite blood-pressure observations, but have depended on the therapeutic test for diagnosis. Thus a dentist, aged about forty-five years, whose large practice necessitated his standing by his chair throughout the day, complained of continued exhaustion which compelled him to take frequent vacations. When he adopted an abdominal belt exerting firm pressure over the lower half of his abdomen he experienced a return of vigor which enabled him to do his work with comparative ease. In the group of complaining, more or less healthy-looking women in whom it is so difficult to localize the pathological condition, and which the physician commonly characterizes as "neurasthenic," measures taken to support the abdominal circulation, often seem to give better results than any other, whether or not there is evidence of enteroptosis. A patient, aged sixty years, who frequently feels an approach of faintness when driving his motor car, has learned that he can quickly restore himself to normal by a series of vigorous contractions of the abdominal muscles. The effect, of course, is to unload the abdominal circulation as by massage.

The one physical sign which seems to be indicative of general, and especially of intracranial, hydrostatic circulation deficiency is to be found in the postural changes of the blood-pressures.

Study of a relatively small number of healthy subjects leads me to agree with the majority of observers in this field, that in the normal person in the erect, at least in the sitting, posture the blood-pressures are higher than when recumbent.

When I find the blood-pressures, especially the maximal, higher in the recumbent than in the sitting posture I conclude there is physiological weakness either of the splanchnic vasomotor system, of the abdominal wall or both and that potential cerebral anemia and vasomotor overstrain are consequences in the erect posture.

In studying a series of cases it becomes obvious that there is no strict parallelism between the acuteness of the symptoms and the difference in the postural heights of blood-pressure. It is probable that, as illustrated in the first case recorded above, the urgency of the symptoms is due to nervous efforts to compensate for hydrostatic failure of the circulation.

In persons employed in earning their living at more or less sedentary occupations, whom through years it has been difficult to keep keyed up to their work, I have learned to expect a higher recumbent blood-pressure. When there is cardiac degeneration a considerable excess in the blood-pressure when recumbent is, in my experience, a bad prognostic sign.

Having been led by such experiences as have been detailed to suspect that splanchnic stasis is a potent cause of many of the disabilities, such as weakness, headache, dizziness, for which the physician is consulted and an important factor in many specific disorders, it seemed to me desirable to study the blood-pressures in their postural relations, with a view to establishing a rational basis of treatment.

My observations were made with a Pilling sphygmomanometer. With the patient sitting the broad cuff was applied around the bare arm, the forearm resting upon a table. At least three estimations of maximal and minimal pressures were made by the auscultatory method. Without detaching the cuff the patient was then made to lie on a couch and the observations were repeated, care being taken to preserve the relative position of cuff to heart.

The diastolic pressure was assumed to correspond with the beginning of the "fourth phase" of sound from the relaxing artery.

In carrying out such work with people physically below par it is astonishing how variable are the individual sound phases both as to their occurrence and intensity.

If these observations were to be repeated I would choose the beginning of the fifth phase, or the cessation of sound, as the point of minimal pressure.

A curious manifestation, apparently significant of functional nervous instability, is not infrequently witnessed in a rapid change in the height of maximal arterial pressure in consecutive observations under constant conditions. The minimal pressure takes an insignificant part in these fluctuations. They seem to be due to subconscious mental perturbations. Nervous women with hypertension at and beyond the menopause are especially apt to show the vasomotor instability. In these cases, in a series of consecutive observations the maximal pressure is prone to be considerably higher in the early than in the late measurements. It is significant that these wide differences in maximal pressures, amounting to 10 mm. Hg. or more, seem to occur only in the sitting posture, in which the vasomotor effort must be most active.

Goodman and Howell[16] have performed a useful service in calling attention to the relation which they claim to exist between the duration of the sound phases and cardiac efficiency. The duration of each sound phase is indicated by the number of millimeters fall which the mercurial column suffers within that phase and the ratio

[16] Amer. Jour. Med. Sci., 1911, cxlii, 334.

which this bears to the pulse pressure is determined. Increase in the second and third phases is held to be a sign of cardiac strength; of the first and fourth of cardiac weakness; the ratio of the two is then easily determined. Swan[17] used the method in the study of a considerable number of clinical subjects and believes it to give a valuable index of the ratio of cardiac strength to cardiac weakness.

The subject is mentioned here because my observations indicate that postural changes which redistribute the blood have marked influence on the existence and prominence of the sound phases and, as already said, circulation efficiency is measured by the integration of conditions throughout the vascular tract.

Very few of the patients to be considered were the subjects of cardiac or vascular disease. None was confined to bed. A few had long ceased to need medical care.

The subjects have been divided into two groups. The first group is composed of those whose systolic blood-pressure in the sitting posture is equal to or greater than that in the recumbent posture. The cases number 26 (9 males and 17 females) with ages varying from eight to fifty-five years. The second group is composed of those whose systolic blood-pressure is greatest in the recumbent posture. The cases number 40 (17 males and 23 females) with ages ranging from eleven to seventy-seven years. The averages of the blood-pressures under all conditions are represented in the accompanying table:

AVERAGE BLOOD-PRESSURES IN MM. HG.

	Posture.	Maximum.	Minimum.	P. P.
I. 26 cases	Sitting	123	77	46
	Supine	120	73	47
II. 40 cases	Sitting	116	71	45
	Supine	122	72	50

Occasionally a patient is found who on one occasion will fall within this and on another into that group. It is not expedient to dwell upon the individual clinical characteristics of the two sets of subjects, but it is interesting to observe that though nearly all were sufficiently below par to seek medical advice, there is a rather lower average physiological grade in those in the second group having the higher recumbent maximal blood-pressure.

This conception is of decided practical value, for included in the second group is a number of persons whose main dyscrasia, such as tendency to acidosis, slight tendency to cure in pulmonary tuberculosis, neurasthenia, might easily depend upon a remedial weakness of the circulation. Perhaps the most significant pressure change in Group II is the rise of the average minimal pressure in the recumbent posture. According to Erlanger and Hooker, there is normally a decided fall in minimal pressure in the recumbent as

[17] Internat. Clinics, 1914, iv, 130.

compared with the erect posture. While in all of Group II the maximal pressure was higher in the recumbent than in the erect posture the minimal pressure was higher in the recumbent posture in 23 cases and lower in 15.

The lowering of minimal pressure with a rise in the maximal on assuming the recumbent posture does not support the conclusions of Pilcher[18] who found, in general, that under all conditions modifying maximal pressure the minimal pressure changed in the same direction if in different degree.

Inherent difficulties impair accuracy in the estimation of the minimal blood-pressure, especially in such debilitated persons as were the subjects of these observations; one or more of the sound phases may be absent or drop out in a change of posture. In such a case the terminal sound was taken to mark the minimal pressure.

My own data seem to indicate that the more normal the subject the more likely is the maximal blood-pressure to fall in changing from the sitting to the recumbent posture, and still more likely is the minimal pressure to fall. Consequently, as suggested by Elanger and Hooker,[19] increase in the pulse pressure in the recumbent as compared with the erect posture is a mark of physiological harmony. Treatment for splanchnic stasis has frequently been instituted on account of clinical symptoms alone; when relief has been experienced by these patients an examination of their postural blood-pressures has always shown them to be included in my second group, that in which the systolic pressure is higher in the reclining posture. The relations of splanchnic stasis to intoxications from the digestive tract seems well worth investigation, but the subject appears to have received little or no attention.

A study of the table from which my averages are made in connection with histories of the patients suggests very definitely that a similar table constructed with adequate care from a large number of subjects would be of prognostic value in the estimation of life insurance risks.

My efficient intern on medical service at the Denver County Hospital, W. K. Hobart, made a prolonged study of the postural blood-pressures in a dozen cases, including 5 with bronchial asthma and chronic bronchitis and 1 convalescent each from lobar pneumonia and typhoid fever. In the pneumonia case, as convalescence became established, the minimal blood-pressure fell markedly in the recumbent posture and the pulse pressure increased correspondingly. In a case of severe chronic nephritis and arteriosclerosis the systolic blood-pressure was usually considerably higher sitting than lying, but the pulse-pressure was reversed from the normal, being higher in the sitting posture. In the, mostly very severe, cases of bronchial asthma all pressures were very variable; four of the

[18] Amer. Jour. Physiol., 1915, xxxviii, 209. [19] Loc. cit.

patients showed at times an extraordinary fall in the pulse-pressure sitting, due to rise in the diastolic pressure. Mere laxness of the abdominal wall, which of itself must tend to splanchnic stasis in the erect posture, may be overcompensated, probably through splanchnic vasomotor spasm, in a most unexpected way. But in such cases I have found that while the maximal blood-pressure might, imitating the normal condition, be higher in the sitting than in the recumbent posture, the value of the minimal pressure, and of the pulse-pressure, would depart more or less widely from the postural variations of health. The general conclusion from the observations is that the postural relations of the blood-pressures, especially as manifested in the fluctuations of the pulse-pressure, are an important index to the state of physiological efficiency.

Treatment of Abdominal Blood Stasis. Oliver,[20] in his monograph states the problem in the treatment of splanchnic stasis to be "to widen the bed of the skeletal arteries, to unload the splanchnic veins, and to tone the splanchnic arterioles." Tension and respiratory and resistance exercises and cycling are advised as especially adapted to gently unload the abdominal veins into the systemic arteries. It is important that each exertion should be followed by a period of rest in the recumbent position. Baths with alternating temperatures, conjoined with percussion and massage which cause dilatation of the systemic vessels have a favorable effect. Oliver points out that the physiological determination of blood to the abdominal viscera during digestion makes it especially advisable that the recumbent position be assumed after each meal. The subject should lie down for an hour at such times, and it is advised that during the latter half of this period a weight of ten to fifteen pounds, conveniently in the form of a bag of shot, be distributed over the abdomen.

These measures seem reasonable, and to them should be added exercises especially adapted to strengthening the abdominal muscles, such as raising the legs or the trunk from the recumbent posture, contraction of the abdominal muscles against resistance, etc.

The rowing machine would seem to be particularly fitted to strengthen the weakened functions. But while our ideal must be correct physiological defects by physiological means, and to strive for it, in medical practice we adopt such measures as relieve morbid symptoms in the quickest way. I know of nothing which so nearly fulfils this purpose in splanchnic stasis as artificial support applied over the lower abdomen.

From the numerous devices heralded for this purpose it is difficult to find one which is at once comfortable to the patient and effective in achieving results. The problem seems easier in the case of women who can wear a corset, laced in front, by which abdominal

[20] Loc. cit.

pressure can be regulated to a nicety without interference with respiratory movement. The fixture—belt, band, or corset—should be adapted to the individual who wears it, and should be regarded as a makeshift during the endeavor to reëstablish correct physiological relations.

SUMMARY AND CONCLUSIONS. This contribution is a plea for the view that splanchnic stasis is not a pathological curiosity, but that it is potentially present, and may be the starting-point for vicious circles of derangement in every case of general functional weakness.

It has been shown that in the erect posture the blood would largely gravitate into the splanchnic veins were it not for the reaction of physiological mechanisms of which the splanchnic vasomotor apparatus and the muscles of the abdominal wall are the chief factors. Laxness of the abdominal wall, probably leads, in the erect posture, to the establishment of a negative pressure within the abdomen, which it is a prime object of treatment to correct.

It is pointed out that depletion of the intracranial blood current must follow insufficient compensation of the hydrostatic pressure involved in the erect posture. Virtual anemia of the brain leading to a multiplicity of disorders, not the least of which is probably vasomotor overstrain, is the natural sequence. It is probable that excess of blood-pressure in the recumbent as compared with the erect posture is a trustworthy index of splanchnic stasis.

No attempt has been made here to exploit the possible influence of splanchnic stasis on the oxygenating power of the lungs or their resistance toward infection or on the metabolism of the abdominal viscera and intoxications arising therefrom.

THE FASTING TREATMENT OF DIABETES MELLITUS, WITH SPECIAL REFERENCE TO ACIDOSIS.[1]

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SINCE its inception by Rollo the dietetic treatment of diabetes mellitus has had as its object the attainment of the aglycosuric state. To attain this state, carbohydrate was withdrawn from the diet; this was done, but never quite satisfactorily, because with-

[1] Read before the New York Academy of Medicine, December 21, 1915.

drawal of carbohydrate from the diet, it was believed, precipitated acidosis. And because it was supposed to precipitate acidosis, prolonged fasting was considered inadvisable, although it was recognized that the occasional fast days of Naunyn diminish acidosis. This procedure was not followed, because it was thought that "fats burn in the fuel of carbohydrate." The spirit of this phrase has dominated the modern treatment of acidosis. The doctrine that overfeeding with fat is harmless and even beneficial in increasing weight and strength and that carbohydrate feeding is required to avert threatening coma has done much to retard the progress of the treatment of diabetes. In point of fact the excess of fat in diet disposes the patient to acidosis; the excess of carbohydrate to glycosuria. Now it has been found that both features of the disease—glycosuria and acidosis—must be recognized and controlled. Both can be influenced by diet; both can be influenced by fasting. In order that treatment by these means may be properly applied it is necessary to understand the effect of diet and fasting upon both glycosuria and acidosis. As far as glycosuria is concerned a few days of complete fast may render the patient sugar-free and a subsequent restriction of diet may suffice to maintain him in this state; but the control of acidosis is more difficult and involves a comprehensive understanding of the problem of acidosis in general. It is, indeed, the presence or absence of this condition which is now regarded as the dominant factor in determining the course of treatment. In view of this fact the present paper is limited to a discussion of this phase of the subject.

Acidosis. By acidosis is meant a decreased alkaline reserve of the blood rather than an increased acidity. An accumulation of acids sufficient to create an acid reaction in the blood and tissues is incompatible with life. The alkalinity of the blood is maintained chiefly by the amount of bicarbonate it contains. This amount may be estimated indirectly in terms of carbon-dioxide tension in the alveolar air, directly by the power of the blood plasma to combine with carbon dioxide. If acids accumulate in the blood and tissues sufficient to neutralize a portion of the bicarbonate there present, they reduce the alkaline reserve of the blood and tissues below normal. When this occurs it is accompanied by a diminution in the power of the blood plasma to combine with carbon dioxide, and consequently causes a parallel diminution in the tension of alveolar carbon dioxide. This condition is known as acidosis, although an acid reaction of the blood never occurs unless *in extremis*. Acidosis, therefore, may be defined as a lessened bicarbonate reserve in the blood and tissues. It may be caused either by an overproduction of acid bodies in metabolism or by a lessened excretion of these substances.

The need of a method to determine the amount of acid bodies in the medium where accumulation takes place is obvious. That

medium is the blood. Tests of the urine are valuable only for the excretory phase of acidosis. They may be misleading in that they give no index of the accumulation of these substances in the blood due to overproduction or to faulty elimination.

Methods for Determining Acidosis. Until recently urinary tests alone have been used as indices of acidosis. These tests have included qualitative tests for acetone and diacetic acid; quantitative determinations of the acetone bodies, of ammonia, and of total acidity. So long as excretion keeps pace with acid formation, tests of this nature are satisfactory; but when excretion of acid substances is imperfect, as may occur in diabetes, the elimination cannot keep pace with production. Therefore urinary tests may not reveal the degree of acidosis present. During the past thirteen years Beddard, Pembrey, and Spriggs[2] have published a series of papers on the amount of carbon dioxide in the blood, the urine, and the alveolar air as criteria of acidosis in diabetes. Since 1913[3] they have abandoned blood analyses in favor of determinations of the alveolar carbon dioxide, because their studies led them to believe that "estimations of alveolar carbon dioxide by Haldane's method are at present the best guide for the prognosis and treatment of diabetes." The alveolar carbon dioxide, being an indirect measure of the bicarbonate content of the blood, is significant, but in abandoning examination of the blood the English investigators have substituted an indirect measure of acidosis for a direct one. The indirect method, namely, the alveolar carbon-dioxide determination, is an accurate indicator of the reserve bicarbonate of the blood only when respiration and circulation behave in an entirely normal manner both as regards mechanical efficiency and nervous control. The fact that Beddard, Pembrey, and Spriggs gave preference to the indirect alveolar-air method, despite its possible inaccuracies, may be attributed to the lack of a satisfactory technic for blood analysis.

Van Slyke's Method for Determining the Bicarbonate Content of the Blood. During the present year a direct and exact method has been introduced by Van Slyke[4] for determining the bicarbonate content of the blood as measured by the power of the blood plasma to combine with carbon dioxide. This method not only eliminates the error of measuring the blood alkalinity by an indirect method but has the added advantages of simplicity, rapidity, and accuracy. It avoids, furthermore, the error due to personal factors. Van Slyke's apparatus consists of a glass pipette in which the plasma is acidified and the bound carbon dioxide is set free. The volume of gas may be read directly from the scale on the instrument or may be expressed in terms of volume per cent. carbon dioxide in the blood by reference to tables with corrections

[2] Lancet, 1903, i, 1366.

[3] Jour. Physiol., 1913, xlviii, x.

[4] Method to be published in the Journal of Biological Chemistry in the near future.

for temperature and barometric pressure. By a further simple calculation the figure obtained may be translated into terms of alveolar carbon dioxide tension.

This method affords a simple clinical means for determining the bicarbonate content of the blood. The blood estimation in conjunction with urinary findings gives an index of the production and elimination of acid bodies, a knowledge of both of which is essential to an interpretation of the clinical picture. In those instances in which an analysis of the blood cannot be made, valuable information may be obtained by determinations of the alveolar air.

Fridericia's Method for Determining Alveolar Carbon Dioxide. In 1914 Fridericia[5] published a modification of an already well-known method for analysis of the alveolar carbon dioxide so simple and so easily applied to clinical use that it merits a brief description. A glass tube with stop-cocks is so arranged that 100 c.c. of expired air can be readily collected for analysis. The sample of air is shaken with an alkaline solution within the glass chamber and all the carbonic acid is absorbed by the alkali so that the amount of gas absorbed may be read directly in percentage from a scale marked on the tube.

Symptoms of Acidosis. The symptoms due to acidosis vary. They range from the mere irritability, mental torpor, and lessened physical alertness of chronic low-grade acidosis through the hyperpnea, drowsiness, and headache of high-grade intoxication to the nausea and vomiting which presages the onset of diabetic coma. A careful study of the manner in which 22 cases have behaved while fasting has suggested that they may be divided into groups of four definite types. Any given case may react differently to fasting on different occasions. This classification is based on the study of acid production and elimination as determined by blood bicarbonate content and urinary acid excretion.

Group I is composed of cases which maintain a normal bicarbonate reserve of the blood throughout the course of their treatment.

In Group II are placed the cases which recover, while fasting, from acid intoxication, sometimes so severe as to verge on coma.

Group III consists of those cases which show a low-grade acidosis as indicated by a subnormal bicarbonate reserve and heightened ammonia excretion.

Group IV includes those cases which develop acidosis while fasting, though previously no evidence of acid intoxication existed.

I. The cases which show no diminution of bicarbonate in the blood during the fasting period are milder diabetics. They are made aglycosuric easily by a few days of fasting and they do not develop untoward symptoms or clinical signs of acidosis during

[5] Berl. klin. Wchnschr., 1914, ii, 1268.

the fasting period. Their carbohydrate tolerance is relatively high and they have the ability to metabolize mixed diets of moderately high caloric value without showing glycosuria. The therapeutic indication in these cases consists in the adaptation of a diet to their tolerance in order that the aglycosuric state may be permanently maintained. (Chart I.)

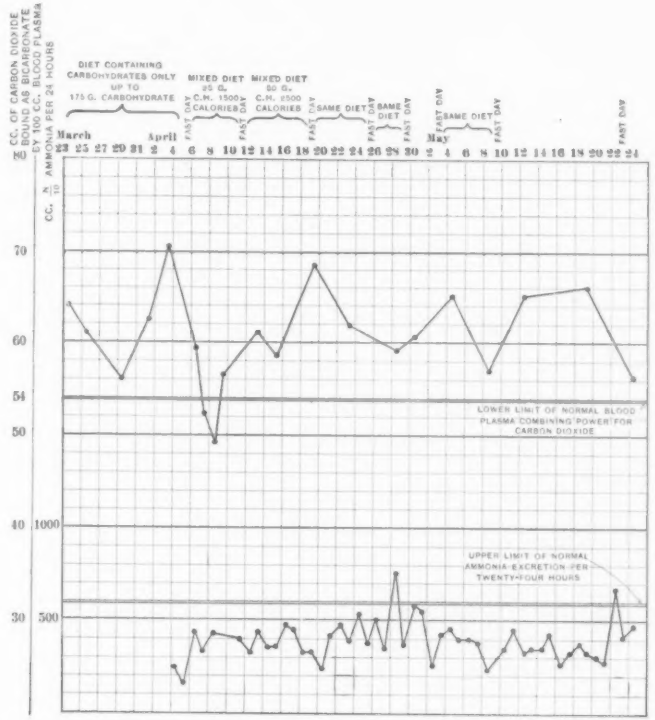

CHART I.—Patient, aged thirty years. Aglycosuric during the period covered by this chart. The power of the blood plasma for combining with carbon dioxide fell below normal only during the period of adjustment following a high carbohydrate diet. The ammonia curve was normal throughout.

II. Group II comprises cases which, during the fast, recover from an acid intoxication which sometimes is so extreme as to verge on coma. These cases are frequently acute in course and develop symptoms of coma with alarming rapidity. There is generally a high glycosuria, a very heavy ketonuria, and a low sodium bicarbonate reserve in the blood (often less than one-half normal). The symptoms in these cases are anorexia, sluggish mental and physical reaction, beginning hyperpnea, and

more or less extreme drowsiness. Fasting is followed by amelioration of all these symptoms. The sodium bicarbonate reserve in the blood rises and generally becomes normal on the third or fourth day, although the urine may not show a lessening in its reaction for ketone bodies until later in the treatment. In these cases the fast should be continued until aglycosuria results. (Chart II.) The patients are then treated by the method in use in this hospital[6] until they remain permanently aglycosuric.

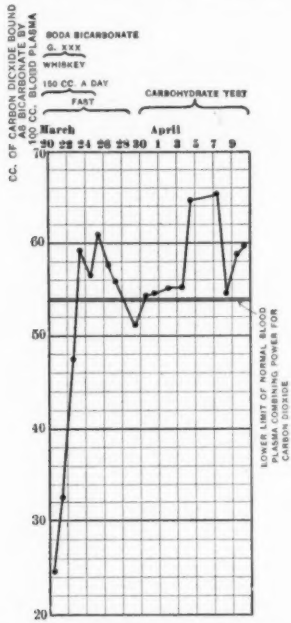

CHART II.—Patient, aged seventeen years. High glycosuria and ketonuria from March 19 to 26. The power of the blood plasma for combining with carbon dioxide rose to normal during the fast, and remained normal after.

III. The cases which show a low-grade chronic acidosis before, during, and after their fast comprise the most difficult group to treat successfully. (Chart III.) One may suspect that a patient belongs in this group from some or all of the following signs:

1. Chronicity of the diabetic symptoms.
2. Lessened mental acumen.
3. Lessened physical alertness.
4. Tendency to a low-grade but continuous glycosuria.

[6] The details of the general and dietetic management employed will be described in a paper to be published later.

5. Low carbohydrate tolerance.
6. A certain degree of obesity.
7. Persistent lipemia.
8. Lessened sodium bicarbonate reserve in the blood
9. A slight but extremely obstinate ketonuria.

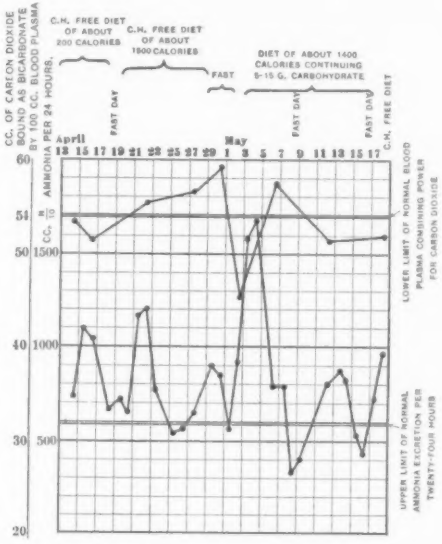
CC. OF CARBON DIOXIDE BOUND AS BICARBONATE BY 100 CC. BLOOD PLASMA
C.H. FREE DIET OF ABOUT 200 CALORIES
C.H. FREE DIET OF ABOUT 1500 CALORIES
FAST
DIET OF ABOUT 1400 CALORIES CONTINUING 9-15 G. CARBOHYDRATE
FAST DAY
FAST DAY
FAST DAY
C.H. FREE DIET
AMMONIA PER 24 HOURS.
April
May
13 15 17 19 21 23 25 27 29 1 3 5 7 9 11 13 15 17
60
54
50
40
30
20
CC. N/10
1500
1000
500
LOWER LIMIT OF NORMAL BLOOD PLASMA COMBINING POWER FOR CARBON DIOXIDE
UPPER LIMIT OF NORMAL AMMONIA EXCRETION PER TWENTY-FOUR HOURS

CHART III.—Patient, aged eighteen years. The patient was observed for fifteen months. This chart is taken from the middle of this period. During the first months of treatment the degree of acidosis was continually higher. At this period and thereafter the acidosis was less marked. Prolonged fasting caused a slight decrease of the power of her blood plasma to combine with carbon dioxide. This increase in the acidosis is not characteristic for the group but is a reaction which may be expected in fasting persons. The ammonia excretion, always high, increased still further to meet the demand for regulation. (These phenomena will be published in detail in another paper.)

These characteristics, as well as the readiness with which the acidosis is increased by fat feeding, suggest that the faulty metabolism of carbohydrate is complicated by difficulty in the complete combustion of fats. The fasting period required to clear up glycosuria may be long. During this period the urine may not become negative in its reaction for ketone bodies, and the bicarbonate content of the blood may maintain its subnormal level. When the aglycosuric condition is reached the patient may still show a low carbohydrate tolerance, rarely more than 60 to 100 grams of carbohydrate in the form of green vegetables alone, and may tolerate only from 0 to 15 grams when allowed a mixed diet of low caloric value (15 to 25 calories per kilogram of body weight).

An excessive supply of fat, even of body fat, together with a low carbohydrate tolerance, apparently plays an important part in the symptomatology of patients in this group, and it is toward the correction of these conditions that treatment should be directed. A continuous subcaloric diet with fast days interpolated twice a week rapidly rids the patient of excessive body fat, while the lowered total caloric intake allows of a greater amount of carbohydrate in the diet without the appearance of glycosuria. Even though the

CHART IV.—Patient, aged twelve years. On admission she had a slight glycosuria and slight ketonuria. During the fast the ketonuria intensified while the glycosuria disappeared, and they both have been absent since. During the fast the degree of acidosis increased to a dangerous extent. The ammonia excretion, though increased, was inadequate. The fast was terminated and acidosis disappeared as measured by the power of the blood plasma to combine with carbon dioxide—the excretion of ammonia and ketone bodies. Two subsequent fasts were attended by no untoward return of these phenomena. (The lower normal level for the power of the blood to combine with carbon dioxide in a child, aged twelve years, should be placed between 50 or 52 instead of at 54.)

loss of weight may appear excessive it is not to be feared. In some instances as much as one-fifth of the body weight has been sacrificed before the clinical, blood, and urinary evidences of acid intoxication have disappeared. Lipemia generally disappears rapidly with the loss of body fat. The patient becomes tolerant of more carbohydrate, the urine loses its reaction for ketone bodies, and the blood gradually rises in its bicarbonate content. Not infrequently months of treatment are necessary to obtain good results in these patients. But persistence, no matter how discouraging treatment

may appear, is almost always rewarded. So far it has been possible to discharge all of these cases on a diet sufficient for their comfort and usefulness and no tendency to a spontaneous return of the former symptoms has been observed.

The cases comprising Group IV are those which develop signs and symptoms of acidosis during the fasting period, though previously no evidence of an acid intoxication may have been shown. (Chart IV.) The hidden danger of impending acidosis may be masked by their apparent state of good health and nutrition. Glycosuria sometimes appears to be the only abnormal factor in an otherwise satisfactory status. Urinary tests may show either a heavy or a negative reaction for ketone bodies. On the second or third day of the fast vague symptoms with irritability may appear. This condition is at first attributed to hunger, but on the following day the symptoms become intensified and nausea may develop or drowsiness supervene. Then the significance of the situation is recognizable. Should fasting be continued still longer, hyperpnea or vomiting, which usually indicates the onset of coma, may occur. During the development of these symptoms, even though the urine may sometimes gradually become clear of glycosuria and the ketone reaction remain slight or only mildly positive, the analysis of the bicarbonate of the blood shows an increasing tendency toward the danger zone. This group emphasizes the necessity of an early recognition of the onset of acidosis and illustrates the advantage of a clinical method which aids in its detection. In these instances acidosis may be manifested when fasting is instituted. The daily analyses of the blood indicate the degree of acidosis and provide the opportunity to control the dangers to which such patients are subjected during the fasting period. A progressive downward tendency of the bicarbonate content of the blood offers a clear-cut therapeutic indication. In these cases fasting should be discontinued. If such a patient is put on a low protein-fat diet the symptoms of acid intoxication may disappear after the first feeding and the bicarbonate content of the blood continues to rise until a normal level is reached and danger of coma is past. After several days of such diet a second fast has always, thus far, been well tolerated and has cleared up the glycosuria successfully.

Twenty-two diabetics have been studied with reference to the effect of prolonged fasting on acidosis. They may be divided into the groups just described, according to their response to fasting as follows:

Group.	Number of cases.	Percentage of cases.
I	10	45.4
II	2	9.1
III	8	36.4
IV	2	9.1
Total	22	100.0

The ten cases (45.4 per cent.) comprising Group I were benefited by the fasting method. They were rendered aglycosuric very rapidly and their stay in the hospital was very materially shortened.

The effect of fasting in the cases of Group II (9.1 per cent. of the series), which show evidence of being in a state of profound acid intoxication, is especially striking. These cases improve, within twenty-four hours, in such a manner that they may be considered out of danger of coma, although they were admitted to the hospital bordering on this state. The two cases cited received sodium bicarbonate (30 grams per diem until the blood bicarbonate reached normal) together with whisky (150 c.c. per diem divided into ten doses). Recently we have treated two additional cases in Group II; they fasted without receiving either bicarbonate of soda or whisky and improved in an equally satisfactory manner. The radical departure in this from previous methods consists in not giving carbohydrate to ward off coma. Certain of our results show that the feeding of carbohydrate in such cases may actually precipitate coma.

The patients comprising Group III (36.4 per cent. of the cases studied) were freed from a chronic low-grade acidosis by prolonged fasting and subsequent continued subcaloric feeding. The temporary undernutrition is not harmful, and temporary loss which is encountered is rapidly made good by improvement. Without the temporary delay, subsequent improvement is often unattainable.

The remaining cases—Group IV—(9.1 per cent. of the series, are those in which serious or fatal results are possible from prolonged fasting if the fasting is not controlled and suitable warning is not heeded. The clinical symptoms alone may not afford sufficient indication of the threatening danger, but the use of the methods described renders fasting safe. Under suitable control, therefore, the fasting treatment can be conducted with safety even in cases of this type.

SUMMARY. The following conclusions may be briefly summarized thus:

I. The object of the fasting treatment of diabetes mellitus is to render the patient permanently free not only from glycosuria but also from acidosis.

II. The term acidosis signifies not an actual acid reaction, but an accumulation of acid bodies in the blood and tissues sufficient to neutralize enough of the bicarbonate there present to reduce the alkaline reserve below normal.

III. Urinary tests for acidosis are often misleading in that they are indicative merely of the excretion of acid bodies and are not a true index of their accumulation. An analysis of the blood bicarbonate as determined by the power of the plasma to combine with carbonic acid, on the other hand, offers an accurate measure of the accumulation of fixed acids and gives a true index of the degree of acidosis present.

IV. For convenience of description the diabetics above described were classified into four groups. This classification is based on the type of response to prolonged fasting as indicated by the degree of blood alkalinity.

V. When the degree of acidosis present is determined daily by the method described, experience so far indicates that all cases of diabetes may be treated by the fasting method safely and with benefit.

THE EAR-TESTS OF BARANY IN LOCATING CEREBELLAR AND OTHER ENCEPHALIC LESIONS.[1]

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PHILADELPHIA, PA.

The importance to the aural surgeon of the Barany tests is generally known, and they have even been overworked in leading to needless operations upon the labyrinth; but in the broader aspects of diagnosis we have probably made only a beginning, and it is to this side of the matter that we would claim attention, particularly the attention of the otologist, who alone can rightly make some of these investigations, since the neurologist and the general surgeon rarely have the needed otological training: and the otologist, for that matter, must renew or enlarge his neurology if he is to progress accurately in this complex field. Yet time for successful intervention in many intracranial morbid conditions is usually short and may not permit of any elaborate coöperative study by a group of specialists—success will be highest when competent head and hand can be united in one person, as we have seen in the low mortality of lateral sinus thrombosis when operated on by the skilled aural surgeon.

With so good a leader as Barany in this modern advance, progress has been rapid and generally sure; yet errors have crept in at many points and obtained temporary currency; and only the combined critical study, tested and corrected by the crucial touchstone of operation or autopsy, can convert the findings from empiric gropings into scientific data leading on to new conquests. For this the large hospitals offer fine opportunities, and we have been very fortunate in Philadelphia to have been able to utilize these to a notable extent.

With this purpose there has been created in the University of Pennsylvania a Department of Neuro-otology, devoted exclusively to this work; and in the past year and a half we have examined

[1] Read before the American Otological Society, June 3, 1916.

125 pathological cases.[2] While giving full heed to the aural aspects, with treatment of any local conditions requiring it, study has been especially directed to the intracranial side of the subject, guided and aided by Drs. Mills, Weisenburg and others on the neurological side and by Dr. C. H. Frazier on the surgical. For this purpose the pointing reactions have far surpassed the nystagmic; and most of our stress will be placed in this paper on these later developments of the Barany testing. Of the intracranial cases eighteen have come to operations and four to autopsy. These sections, antemortem or postmortem, have shown the deductions from the ear-examinations to have been remarkably accurate, and have led us to believe that this work is valuable and worthy of this preliminary report.

As usual in such complex and many-sided researches, anomalous or contradictory findings frequently confront us. Physiological phenomena, not to speak of the psychic, do not group themselves so readily under simple laws as do the merely physical. Yet some inconsistency of findings, even in the same case, marks the vital characteristics of our subject—we are dealing with advancing processes not with fixed morbid lesions; and the variations may be most instructive when understood, however disconcerting they may at first appear to one who vainly undertakes to demonstrate a reaction previously present.

It is hardly necessary at this time to describe in detail the technic of examination. In brief, however, as regards the pointing reactions, the normal person is always aware of the location of his hand or finger in space when his eyes are closed; and he is able with it to find an object previously located by him, as, for example, the finger of the examiner held in front of him. With the ear stimulated, either by cold or hot water or air or by turning in a smoothly revolving chair, he is no longer able to find the finger, but points *past* it in a definite direction, either to one side or the other, above or below, depending upon the exact stimulation employed. These "past-pointing" reactions are absolute in the normal; their modification marks the abnormal and challenges us to find the explanation and locate the fault.

If the right ear is douched with cold water of a temperature of 68° F., the normal person points to the right; if the right ear is douched with hot water of 112° F. he points to the left. This is true, in both instances, also of the wrist, elbow, foot, head, and trunk. Or if the patient is turned in a rotating chair toward the right and then suddenly stopped, all the pointings will be toward the right, and *vice versa* after turning to the left. In each of these tests the head must be rigidly supported in the chosen position.

There has been so much needless confusion, however, in the descriptions of these tests, especially in stating the direction of the nys-

[2] Also many more not found pathological, although we cannot emulate a colleague who claims, "I apply the labyrinth tests to every case coming to the clinic."

tagmus in comparison with the past-pointing, that perhaps we may venture to suggest an absolutely reliable and easy method of remembering these facts. Thus, the usual routine methods are by cold water and turning: now if to the right (that is either turning to the right or douching the right ear) everything is to the right —all the pointings, shoulder from above, shoulder from below, elbow, wrist, neck or trunk, and the falling reactions, all are to the right. Now the deviation of the eyes, just as all the other reactions, is also to the right; but this is the slow movement of the eyes to the right. The "recovery," the quick movement to the left, however, the cerebral component, is more conspicuous, and therefore this phenomenon has been called "nystagmus to the left." *Vice versa*, to emphasize this: if the left ear is douched or the patient is turned to the left, all reactions are to the left and the true vestibular movement of the eyes is also to the left, although mistakenly the nystagmus is spoken of as toward the right. This makes it very simple, especially in teaching students; but to tell them, as does Barany, that the past-pointing is always in a direction opposite to the nystagmus, is not only confusing but misleading; simply tell them—if to the right, everything is to the right; if to the left, everything is to the left.[3] As to the reasons for these reactions:

First as regards the nystagmus, this seems easy and fairly understood. The stimulus travels from the ear to the muscles of the eyes, producing a movement to one side; the quick readjustment in the opposite direction is the cerebral attempt to overcome the abnormal movement. Second, a normal person past-points either to the right or the left, according to the stimulation, because he has a disturbed sensation of his position in space.

Now for a normal person to point, under stimulation, in any direction, there must be a definite arc, just as there must also be a definite tract from the ear to the eye, in order to produce nystagmus. In addition, if douching the ear or rotation produces vertigo, it also naturally follows that there must be an arc from the ear to the subjective centres, which can perhaps be best spoken of as the subjective arc. If, therefore, there should be after stimulation an absence of nystagmus, an absence of past-pointing or an absence of vertigo, we can assume an interruption or lesion along the line of the arc concerned. This is at the basis of this matter of the encephalic localization. The ear is stimulated, the impulse is carried by means of the VIII nerve to a group of nuclei in the lateral part of the medulla, namely the Deiters's nucleus, triangular nucleus, and von Bechterew's nucleus; from here the impulses go in a number of directions. One set goes from the nuclei to the posterior longi-

[3] Is it not time to select a substitute for this misleading word "nystagmus," now hopeless, unless in a general sense, in this connection? In anesthesia or stupor the reaction is solely a conjugate deviation. Why not call it the nystagmic deviation?

tudinal bundle and from here to the VI, IV, and III nuclei in the pons and crus, thus causing a nystagmus; this is definite and in all probability accurate.

As for the cerebellar circuit, it may take many years to work it out positively. Our present conception is that on each side the fibers from the *horizontal* canals go to the Deiters's group, thence to the cerebellum by way of the juxtarestiform body (which is the internal portion of the inferior cerebellar peduncle) through to the cerebellar nuclei, globosus, fastigii, and emboliformis, and then to the cortex of both sides. The fibers of the *vertical* canals apparently have an entirely different course, going directly to the posterior longitudinal bundle, probably entering the cerebellum through its middle peduncle. Another group of fibers undoubtedly goes to the spinal cord.

We have come to a definite conclusion that all pointing reactions are primarily *cerebral* and not cerebellar, as generally stated. When one of ourselves is tested and, for example, with erect head is turned to the right, he has on stopping a subjective sensation of turning to the left. For this reason he feels that he is leaving the finger of the examiner behind him and points to the right, where he conceives the finger now to be. He deliberately points to the right unless by a non-automatic, calculated correction he overcomes the misconception of the position of the finger. If after the usual past-pointing the examiner's finger is moved out to meet that of the examined, who then repeats the test, he past-points almost as much farther to the right, and would continue perhaps to the completion of an entire circle in his chase of the seemingly moving finger but for the limitations enforced by his seating. Yet if rotated and asked to make the same movements of the arm without any finger to touch, he maintains it in the same body-plane with little or no deviation, since he conceives this plane to rotate with his seemingly revolving body. There is, therefore, no cerebellar or automatic drawing of the arm to one side; the past-pointing is purely cerebral. Yet we have it achieved through the synergising action of the cerebellum: with the cerebral mandate is associated the cerebellar influence, the muscle-sense, arthrodial sense, tactile, auditory, and visual impressions and memory. In fact, the pointing reactions are dependent on so many things that it is necessary to assume a complicated arc to be able to explain them.

The movement, therefore, is initiated by the cerebrum, and whatever deviation occurs is because the ear-stimulus has given by artificial means a false conception of the position in space. If we postulate that the sense of direction of the right arm is determined by a stimulus going from the ear to that particular centre of the cerebellar cortex governing the right arm, and from there through the superior peduncle of the cerebellum to the cerebral cortex, and similarly an entirely separate tract of fibers for the left

arm centre, we then can explain why any disturbance along the line of the right arm tract will cause a lack of past-pointing of that arm alone, whereas all other parts would past-point because their circuits are intact.

In conclusion, we can claim that by these valuable ear-tests we can usually differentiate lesions of the labyrinth from those of the cerebellum; we can always tell when the VIII nerve is diseased; we can say positively also whether or not the posterior longitudinal bundle is affected; and we can detect a lesion of the cerebellum but cannot always locate it.

We offer here two contributions that so far as we know are new:

1. The subjective circuit. We postulate this circuit *through the cerebellum* to the higher centres and in proof cite: (*a*) two patients who had perfect nystagmic reaction but no past-pointing and no vertigo; (*b*) several cases of spontaneous nystagmus with no vertigo; (*c*) in most cases of cerebellar lesion dizziness is either absent or subnormal; (*d*) a patient with no nystagmic reaction and yet normal past-pointing and vertigo. As the two latter reactions seem to go together, it may be concluded, at least for the time being, that the impulses which are concerned with the vertigo pass through the cerebellum. Also, arguing backward, if no vertigo is obtained it may prove that there is a lesion of the cerebellum.

2. Our second postulate is that the fibers from the different semicircular canals have entirely *separate tracts* of their own; the horizontal canal has an entirely different set from the tracts for the superior canal. This we have proved in 28 cases by means of the following phenomena: In all of these there was little reason to doubt that the region of the posterior longitudinal bundle was implicated. In each of these cases douching of the superior canal (that is with the head upright) produced neither nystagmus, past-pointing, nor vertigo; whereas on stimulation of the horizontal canal (with the head back 90°) all the reactions came through promptly—nystagmus, past-pointing, and vertigo. It is therefore evident that there is a complete central differentiation for the different canals of all three tracts, remembering that in none of these cases was there a peripheral lesion. Autopsy or other findings in some of these cases with no involvement of the horizontal canal fibers of either side, yet with no passing of the stimulus by the superior canal fibers, showed that in the medulla and pons the fibers for the superior canals are mesially located, whereas the horizontal canal tracts are external.

Further details might be given as showing the possibilities of scientific research in these lines, namely, the finding and defining of encephalic tracts by means of these ear-tests. Furthermore, it is not improbable that we shall be able to locate in the trunk of the VIII nerve more definitely than we have yet done the relative

positions of these bundles of fibers, as has been partly done with much value in the optic nerve. Just as the older anatomists recognized that the "portio molis" and "portio dura" could be wholly differentiated into the acoustic and the facial nerves of different origin and function, and as later research is separating the cochleal from the vestibular and intermediary nerves, so it is our part more minutely to differentiate these trunks into their components and by tracing their encephalic course to improve our diagnostic possibilities, with further practical refinements in localization. To this good task we invite the aid of all our colleagues.

FURTHER EXPERIMENTATION IN ANIMALS WITH A MONILIA COMMONLY FOUND IN SPRUE.[1]

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In a paper read before the Association of American Physicians May 13, 1915, entitled "Studies in Moniliasis of the Digestive Tract in Porto Rico,"[2] and in one read before the Medical Society of the District of the North of Porto Rico, March 18, 1915,[3] reference is made to incomplete results of animal experimentation with a new monilia found in sprue, promising additional observations. This paper is in continuance of these experiments, and although experimental moniliasis in animals is yet in its infancy, nevertheless, some striking results in a short series seem to warrant prompt mention, the writer realizing, however, that these rapid preliminary investigations should only be understood as mapping out future and more elaborate lines of study.

As will be seen by reference to the above-cited papers and others of recent date, "Is Sprue a Moniliasis of the Digestive Tract,"[4] and "The Dietetic Treatment of Sprue,"[5] there is justification for considering Bahr's opinion that sprue is due to a monilia as correct, as well as for admitting, as will be seen, that this organism is not Monilia albicans, at least in Porto Rico, but as far as can be determined a distinct and heretofore undescribed species, which I have

[1] Read by title before the Academy of Medicine of Porto Rico.
[2] Am. Jour. Med. Sci., 1915, cl, 680.
[3] Jour. Am. Med. Assn., 1915, lxiv, 1893.
[4] Am. Jour. Trop. Dis. and Prev. Med., 1915, iii, 32.
[5] Now being published by Am. Jour. Trop. Dis. and Prev. Med.

found in now nearly 100 cases of true sprue and in but a small percentage of carriers.

All work upon animals up to date will be succinctly stated, and finally tentative deductions will be drawn therefrom.

Hypodermic Inoculations. *Rabbit I.* Received on December 26, 1914, five drops of a seven-day glucose bouillon culture, 2 per cent. plus 1, in the muscles of the tongue. This was a pure culture from Case IV, isolated in November, 1914, from the inflamed tongue of a typical case of severe sprue whose history was chronicled in "Clinical Notes on a Case of Sprue."[6] This case, thought to be recovering, relapsed and died in the United States early in 1915. The diagnosis was not only confirmed by consulting physicians here in Porto Rico, but was independently made in New York by Crow, of the U. S. Navy, and tacitly accepted as sprue by the *Tropical Diseases Bulletin of London*, March 15, 1915, No. 4, iv, 225. This strain, therefore, has been used in all save one or two instances in animal experimentation, and culturally and morphologically is identical with all others isolated as Monilia X from my cases of sprue.

In the operation of hypodermic injection of the dose into the tongue the animal received also about 2 c.c. per oram. After forty hours diarrhea began and the animal was seized with great trembling, falling about as though intoxicated. At times it would scream out and then lapse into a semicomatose condition, its snout gradually falling to the floor of the cage, upon which contact it would endeavor to straighten up and brace itself in a sitting posture. Much abdominal gas was developed and the mucous membranes were purplish. Death in seventy-five hours.

Autopsy. Tongue presents a dark congested spot at the site of injection, which revealed in section a necrotic area, surrounded by a wide zone of inflammation.

Stomach shows a white grumous coating filled with yeasts. Small intestine likewise so coated, and entire intestine full of gas. Right heart filled with blood. Lungs highly congested, as were liver and spleen. Kidneys large and congested, and all save heart were friable, punctuated, on section, by minute white dots.

Pure culture of Monilia X directly obtained from the following organs: spleen, liver, kidneys, lungs and heart's blood, no contaminating organism being found. Also from the necrotic spot in the tongue, and the mucous membrane of the stomach and small intestine. Cultures from the surface of the tongue were negative for yeasts.

Microscopically the liver and kidneys were examined, the former showing an occasional yeast; the latter demonstrated large embolilike colonies in the cortex, with surrounding zone of intense inflam-

[6] Bol. Assoc. Med. de Puerto Rico, 1914, x, 10.

mation. It is interesting to note the prevalence of short hyphæ and oval forms in the kidney. Stain: Gram-Weigert fibrin method and Bodin's victoria blue and erythrosine. As these emboli-like colonies in the kidneys were thus identified as "the minute white spots" mentioned in the gross anatomy of organs of animals dying after infection by monilia it is presumable that all these necrotic areas were similar.

After five months, during which the original culture had become old and full of mycelium, and had also been several times replanted, Rabbit II (A and B) received 5 c.c. each of a thirty-day culture in sugar-free bouillon of Monilia X from same case (IV) by intraperitoneal injection. No effect whatsoever after two weeks, save possible temporary intoxication upon injecting the dose. This appeared in a half hour, and had disappeared in about three hours. Rabbit II (B) received four or five consecutive inoculations of the same culture in 10 c.c. doses without effect, save the temporary intoxication above noted. Despairing of a successful inoculation of these two animals intraperitoneally, I injected 5 c.c. of a week-old culture in glucose bouillon into the connective tissue of the ear of Rabbit II A. My original intention was to give an intravenous injection, but the squirming and kicking of the animal caused me to discharge the dose instead into the connective tissue. There was no effect whatsoever upon the rabbit's apparent general health, but within three weeks the ear became the site of a huge "blastomycotic ulcer," which slowly healed of itself.

White Rat I. Repeatedly injected intraperitoneally with 1 c.c. of the thirty-day culture of Case IV as in the case of Rabbit II (A and B). No effect after three weeks' observation. He then received 4 c.c. of a five-day culture in glucose bouillon, 2 per cent. plus 1 from Case XV, a severe and typical case of sprue, originally recovered from the patient but five weeks before, and after having been passed through Rat III. Death in eighteen hours.

Autopsy. Lungs highly congested. Liver, spleen, and kidneys dark red and friable. No peritonitis. Cultures from all organs positive for Monilia X.

White Rat III. Received intraperitoneally 5 c.c. of a ten-day culture in saccharose bouillon of Monilia X from Case XV. Died in the night, five days later. An interesting feature of this case was that the site of injection necrosed, exposing the intestines over a space 1.5 cm. in diameter.

Autopsy next morning. Rigor mortis marked. Ulceration of abdominal wall over an area 1.5 cm. in diameter, leaving a perforation communicating with the interior of the peritoneal cavity. Lung deeply congested; spleen small; liver congested. Cultures from spleen and liver positive for Monilia X.

Guinea-pig II. Received intraperitoneally 1 c.c. of a glucose bouillon culture, 2 per cent. plus 1, of Monilia X recovered from

Case XCII (*a*), a severe advanced case of sprue. Daily injections were given beginning August 19. Cultures for first three injections were one week old. Thereafter from August 25 fresh cultures were substituted: the first twenty-four hours old; the next forty-eight hours; and so on until, on August 31, the animal died, having received that day 1 c.c. of a 168-hour culture. Death in twelve days.

Autopsy. Performed immediately. Lungs highly congested with minute white spots. From all organs typical colonies of Monilia X.

Guinea-pig III. Received intraperitoneally 1 c.c. of a forty-eight-hour glucose bouillon culture, 2 per cent. plus 1, recovered from Guinea-pig II. Death in forty hours.

Autopsy. Performed immediately. Only gross changes found were high congestion of lungs, spleen, and liver, which were friable and filled with minute white nodules. Cultures from lung, liver, and heart's blood typical of Monilia X. Cultures of a monilia recovered from the excreta were atypical.

Guinea-pig IV. This animal received 1 and 2 c.c. doses, intraperitoneally, of the old non-virulent cultures from Case IV, used unsuccessfully on Rabbit II, A and B, and White Rat I, as above detailed. By this time the culture, however, had been growing for nearly a year in Sabouraud's glucose agar, 4 per cent. plus 2, with monthly transplantations. The cultures injected were a week old in glucose bouillon, and were given daily for nine days. On the tenth day 5 c.c. were injected and the animal died the next morning.

Autopsy. Immediate. Usual macroscopic findings. The original monilia was recovered from the peritoneum, diaphragm, spleen, adrenals, and mucous membrane of the bladder.

Guinea-pig V. Received intraperitoneally 1 c.c. of a forty-eight-hour culture of the organism recovered from Guinea-pig IV. Death in ten hours.

Autopsy. Lungs, liver, spleen, and kidneys highly congested with white spots. Peritoneum normal. Typical colonies of Monilia X recovered from kidney, spleen, heart's blood, and lung. Peritoneal fluid negative for monilia. The organism recovered from this animal killed animals after ingestion of its cultures, as will be seen later.

Guinea-pig IX. Received 1 c.c., intraperitoneally of a six-day glucose bouillon culture, 4 per cent. plus 1, obtained by culture from kidney of Guinea-pig V. Time to kill not known, but was within twenty-four hours.

Autopsy. Died during night and autopsied next morning. The only thing of note was a high degree of congestion of lungs with its corresponding minute white spots. No peritonitis. Cultures from liver, spleen, and kidney gave colonies typical of Monilia X. Heart's blood and lung negative.

Guinea-pig X. Received intraperitoneally 1 c.c. of a six-day glucose bouillon culture, 4 per cent. plus 1, of Monilia X recovered

from Guinea-pig III (Case XCII (*a*)). Died within twenty-four hours in convulsions.

Autopsy. Hemorrhagic fluid in peritoneum. Lung highly congested with minute white spots. Cultures from kidney, heart's blood, liver, spleen, and lung positive for Monilia X.

Guinea-pig XII (*b*). Received intraperitoneally 1 c.c. of a glucose bouillon culture, 4 per cent. plus 1, of Monilia X recovered from Guinea-pig VIII, which had died after ten days' feeding on culture of the monilia recovered from Guinea-pig V. This experiment was primarily made to sustain the virulence of the strain recovered from autopsy of Guinea-pig VIII. Died in seven hours.

Autopsy. Immediate. Considerable increase in peritoneal fluid. Bladder full of urine, which has a heavy sediment. Lung highly congested with white points. All abdominal organs friable and dark red. Cultures from spleen, liver, urine, kidney, peritoneum, stomach, and lung were made, but only liver, lung, and peritoneal fluid were positive for Monilia X.

Guinea-pig XII (*a*). Same as above, but died in twenty hours. Culture from spleen positive for Monilia X.

Guinea-pig XVII. By intraperitoneal injection of 1 c.c. of a five-day glucose bouillon culture, 4 per cent. plus 1, of Monilia X recovered from the spleen of Guinea-pig XII (*a*). Death in nineteen hours.

Autopsy. Increase in peritoneal fluid; bladder full of urine; much gas in intestines; friable, dark red abdominal organs; minute white spots in liver, spleen, and lungs. Testicles highly inflamed. Cultures from peritoneal fluid, liver, spleen and testicles positive for Monilia X.

FEEDING EXPERIMENTS. *Guinea-pig I.* On the strength of advice given by French authors that animals may only be made susceptible to infection by certain yeasts in a condition of reduced vitality from underfeeding, this animal was fed for a week on a patent malted baby food alone, a food to which it was foreign, and one offering excellent pabulum for monilia. Beginning January 8, 1915, 10 c.c. of a fresh bouillon culture of Monilia X from Case IV was daily mixed with this food. Within three days he became very ill and seemed about to die. Had diarrhea and was somnolent. Death on January 25, 1915, seventeen days after the feeding with cultures was begun.

Autopsy. Performed eight hours after death. Animal somewhat emaciated. Tip of tongue pink but no mouth lesions. General glandular enlargement, chiefly of submaxillary glands, but the axillary and inguinal glands were also increased in size. Section of all glands showed a high degree of inflammation. Liver and spleen were highly congested and friable. Lungs and kidneys were the same, but to a less extent. Stomach and small intestine were covered with a creamy, grumous coat; stomach congested in spots; intestines highly congested. Bladder filled with urine.

Peritoneum normal. Cultures from lung, cervical and inguinal glands, small intestine, heart's blood, kidney, and liver were all negative. Cultures from spleen, tongue, and stomach gave colonies of monilia. All save culture from the spleen were identical with Monilia X.

Guinea-pig VIII. In the case of this animal it was determined to depend entirely upon a hypervirulent monilia, previously passed, as has been seen, through Guinea-pigs IV and V, and to allow the pig to eat its malojillo grass undisturbed by the addition of any carbohydrate. These cultures were merely poured over the grass. 10 c.c. per diem of a seven-day glucose bouillon culture of the monilia recovered from Guinea-pig V was thus administered. On each subsequent day the culture was one day older, as all were sown the same day. The absence of Monilia X from the feces was previously determined. Feeding began September 17, 1915. On September 29 the animal was very sick, with hair on end, abdomen distended with gas, and lips and nose fiery red and raw. The animal died during the night.

Autopsy. September 30, 10 A.M. Animal slightly emaciated. All around the borders of the mouth, especially at the angles and inside the lips, the mucous membrane was red and raw. The entire lower portion of the nose was raw and blood issued from both nostrils. Raw area on the inside of the right cheek. From the upper border of the lip to half an inch above the level of the nostrils and for one-eighth inch external to same, denuded. Tongue apparently normal. The subcutaneous tissues of the animal were congested from the level of the ensiform appendix upward, this being especially noticeable in the neck. The submaxillary and cervical glands were highly inflamed, congested, and swollen, and of a dark red color. Lung highly congested, especially posteriorly, and was filled with minute white spots. The liver and spleen were dark red and friable, the liver being enlarged. The kidneys were twice their natural size and dark red. Peritoneum apparently normal, but there was an excess of blood-tinged peritoneal fluid, without adhesions or pus. Bladder filled with urine. Stomach and intestines a lively pink. Lower bowel normal.

Cultures from the excoriated mouth and nose, small intestine, stomach, tongue, esophagus, and lung were positive for Monilia X. Submaxillary gland, lumen of the trachea, cervical glands, kidney, heart's blood, peritoneal fluid, liver, and urine in bladder were negative.

Guinea-pig XI. Six healthy guinea-pigs were chosen, and one other which had been partially immunized by six small doses, injected intraperitoneally of a virulent culture of Monilia X. They were all placed under the same conditions in the same division of a cage. In the adjoining division, separated by a wooden partition for half the height of the cage and wire netting for the upper half, were other healthy control pigs. The seven experimental animals

received upon their ration of malojillo grass 48 c.c. of a six-day glucose bouillon culture of Monilia X recovered from Guinea-pig VIII. This was poured over their conjoint food, a dose of 7 c.c. to each a day. These feeding experiments began October 6, 1915. On October 7 the smallest pig (XII A) died.

Autopsy. Immediate. One roseolous spot on the upper lip. The submaxillary glands were swollen and inflamed. No peritonitis, but increase in peritoneal fluid. Bladder full of urine. Large intestine full of a fermented liquid and distended with gas. Lung highly congested, and this organ and the spleen showed minute white spots. Kidneys and liver congested and enlarged.

Cultures from kidney, lip at site of inflammatory spot, tongue, heart's blood, stomach, spleen, and large intestine positive for Monilia X. Contents of gall-bladder and peritoneum were negative.

The rapid death of this animal after a day's feeding naturally aroused my suspicion that it had fallen a victim to an epizoötic at times observed among guinea-pigs here and chronicled in standard works, but the prompt demonstration of Monilia X in the above-mentioned organs caused me to attribute its death to the organism upon which it had fed.

Guinea-pig XI B. Died October 8, forty-eight hours after beginning experiment.

Autopsy. Increase in peritoneal fluid, which is of a citrine color. Bladder full of urine. Intestine very full of gas. Lung congested with minute white spots. Solid organs of abdominal cavity dark red and friable. Cultures from tongue and lung positive.

Guinea-pig XI C. Died October 12, six days after beginning the experiment.

Autopsy. Increase in peritoneal fluid. Bladder full of urine. Intestine full of gas. Lung congested with minute white spots. Solid organs in abdominal cavity dark red and friable. Cultures from tongue, lung, stomach and liver positive.

Guinea-pig XI D. Died October 13, seven days after beginning experiment. Autopsy same as for Guinea-pig XI C. Cultures from tongue and liver positive for Monilia X.

Guinea-pig XI E. Died October 13, seven days after beginning experiment. Autopsy same as C. Cultures positive from several organs for Monilia X, but record was lost.

Guinea-pig XI F. Still living.

Guinea-pig XI G. This pig had received intraperitoneally, beginning September 17, 1 c.c. of a glucose-bouillon culture from the spleen of Guinea-pig V. The culture was thirty-six hours old when first injected, and 1 c.c. was given daily for six days; the culture being, of course, one day older at each injection, as the six tubes were sown the same day. The animal sickened but did not die. Feeding began, as has been seen, on October 6, and the animal died October 20.

Autopsy. Nothing of note. Cultures were negative from all organs save the stomach and intestines, which were positive for Monilia X.

Monkey II. A small brown monkey was subjected to a preliminary diet similar to Guinea-pig I for a week, then to this food was added 10 c.c. daily of fresh glucose-bouillon cultures of Monilia X from Case IV. The animal promptly began to suffer from diarrhea a few days thereafter, and continued to have very frequent pultaceous, gray movements with much gas during the six weeks the feeding was kept up, and for some weeks after the rigid diet was suspended, and he was given his normal food without the admixture of Monilia X. During all this time he steadily lost in weight, became quite inactive, and seemed quite sick. He eventually recovered.

Monkey III. Large black monkey, weighing four pounds before beginning experiment, who had lived in a cage near my laboratory table for a year, having thus been under my constant daily inspection. This animal had never had diarrhea, and had always seemed healthy. His stools had been sown for monilia, and always yielded a completely different species, culturally and morphologically, from Monilia X. On October 4, 10 c.c. of a six-day glucose bouillon culture from Guinea-pig VIII was twice daily mixed with his normal food. Forty-eight hours afterward he began to have diarrhea, which rapidly became severe, and has so continued to date (six weeks), with intervals of a day or two of respite. In these six weeks the animal has lost one and a half pounds in weight, but he is usually active only occasionally seeming to be sick. He has had no mouth lesions nor excess of gas. His feces are loaded with Monilia X.

OBSERVATIONS. 1. The species of monilia recovered by me from now nearly one hundred cases of sprue, and apparently a new species from careful cultural and morphological investigations, is a pathogen for current laboratory animals by hypodermic inoculation.

2. This species, which I will for the present call Monilia X, is ordinarily a low virulence organism.

3. When recovered from a patient with sprue and promptly injected into certain laboratory animals it generally produces their rapid death from a mycotic septicemia.

4. When grown for a long time and frequently transplanted the same organism which rapidly killed soon after isolation from a sprue patient seems to partially or completely lose its virulence.

5. This virulence may be recovered by passage through susceptible animals, and even reach such a point as to sicken or kill these animals by continued feeding.

6. Ordinarily an animal may not be killed by feeding Monilia X when first isolated from a sprue patient. Its virulence must be augmented by passage before uniform pathogenicity through feeding can be demonstrated.

7. In such animals the symptoms depend on the part of the intestinal tube must affected, in the portion in which these monilia secure their first foothold.

8. A certain number of animals exposed by feeding, rapidly die of a monilia septicemia believed by me to be due to a sudden primary pneumonia and secondary septicemia.

9. Animals escaping this fate succumb more slowly to what seems to be a toxin developed in the intestinal tract by a localization of these monilia.

10. Feeding experiments have resulted in the production of a stomatitis on two occasions and in the appearance of severe and long-continued diarrhea in several occasions.

11. Monilia septicemia causes the necrotic areas in animal organs referred to as "white spots" macroscopically. This has been verified microscopically in kidneys, and presumed to be the same for all organs so affected. Such organs are highly congested, dark red, and friable.

12. Localized in the skin, typical blastomycotic ulcers are formed whose characteristic is necrosis without pus formation. If an internal organ is attacked large colonies of monilia are seen which look like emboli. Intervening spaces are generally free from the organism. This explains a casual failure to obtain a successful culture from an evidently infected organ, and thus differs radically from a bacterial septicemia.

13. Microscopically, infected organs show large clumps of monilia surrounded by an intense inflammatory zone. I have never seen pus produced by monilia.

14. In one guinea-pig presenting a severe stomatitis, sections of the affected zone revealed monilia in the midst of the muscular bundles below the subepithelial connective tissue. This may explain the tendency for sprue to recur after an apparent dietetic cure: the yeasts are starved out in the surface; the patient apparently recovers, and, later, the deep lying roots of the mycelia layer push out toward the surface and reëstablish a surface growth with its consequences and a relapse.

15. In experimental animals in whom mycotic septicemia is induced by intraperitoneal injection the lungs are the most grossly affected; after them the kidneys and microscopic examination corroborate the gross anatomy.

A forthcoming monograph is contemplated with detailed description of morphological and cultural studies of this organism, and illustrations drawn by the artists of the Institute.

My acknowledgements are made to Drs. W. W. King and González Martínez for valuable assistance in autopsies, and to Messrs. José Loubriel and Damián Artau, Technical Assistants of the Institute, for assiduous care of animals and assistance in autopsies.

THE PRESENT STATUS OF THE ELECTROCARDIOGRAPHIC METHOD IN CLINICAL MEDICINE.*

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A GALVANOMETER suitable for taking clinical electrocardiograms was designed by Einthoven in 1903. Nine years ago (1906) the method was perfected and became available for general use. The first papers dealing with the results of clinical electrocardiography appeared six and seven years ago (1908 and 1909). The first American papers were published in 1910. This report, then, records the results of observations extending over a period of less than ten years.

The electrocardiogram is a record of the electrical discharges from the heart during its activity. Its chief merit consists in the fact that its form remains constant from a time shortly after birth until the alterations due to old age set in. If it changes at all, it does so as the result of alterations within the heart, or in the position of the heart within the body. An alteration from the accustomed form therefore represents a change which must be looked upon as significant.

A normal electrocardiogram in reality does not exist. A group of normal persons in the prime of life yields electrocardiograms which vary only within certain limits. They tend, naturally, to resemble one another, and from this circumstance an ideal form, regarded as the normal electrocardiogram, has been evolved. The constant form of electrocardiograms depends in part on the fact that the method of obtaining records is free from errors due to personal equation. If electrocardiograms are taken with a standard instrument, in a standard way, they may be made anywhere by anybody, and should yield identical curves.

The constancy of an individual electrocardiogram, then, is the basis of the method's importance. Compared with any other form of graphic record of circulatory phenomena, this advantage is manifest. No other one is so simple to read. It can be taken without difficulty at any period of life, and without any discomfort to a patient. It cannot, of course, displace other graphic methods when these record activities other than those given in the electrocardiogram. But where information is required which can be obtained by two methods, the electrocardiographic and another, the electrocardiographic is simpler and more reliable.

* Read before the Association of American Physicians, Washington, May, 1915.

The well-established form of the so-called normal electrocardiogram is the basis of the value of the abnormal one. The normal one depends on the orderly performance of a number of functions. An abnormal one results when the orderly performance of one or more of these functions is no longer possible. Four main cardiac functions on which the normal curve depends can now be recognized. In the first place, it depends on the orderly *passage* of impulses, or more correctly on the orderly spread of electrically excited states through each pair of cardiac cavities, and on the duration of such states in each part of the chambers. It should be noticed that the *production* of impulses cannot be detected in the electrocardiogram, but only the electrical states which the activity of the impulses has initiated. In the second place, the normal electrocardiogram depends on the orderly *sequence* of contraction* of the auricles and ventricles. In the third place, it depends on the orderly *coördination* of the muscular mass in each pair of cavities, and finally on the usual *disposition and volume* of the muscular mass of each pair of cavities. The normal electrocardiogram probably depends on other factors besides these, but these are the ones which we can recognize in the records. The orderly performance of these functions is recognized by the accustomed form and by the time relation of the waves. The interpretation of functional alterations in the heart is based, therefore, on the alterations which are seen in the electrical elements (*P*, *R* and *T* waves) which represent them.

I take up in turn the factors which determine the normal electrocardiogram and will enumerate what disturbances in cardiac function the curves permit us to detect. The first function mentioned was the normal *passage of the impulse* in each pair of cavities. When the passage is normal through the *auricles*, it is represented (Fig. 1) by a wave (*P*) having a definite rounded form, a certain duration, and an upward direction.[1] [2] The rate of the heart is shown by the frequency of its regular recurrence. The passage of an impulse having an abnormal origin is detected by an alteration in the form of the wave.[3] The most extreme change is an inversion (Figs. 2 and 3).[4] It is supposed to denote that the impulse has arisen at a lower level of the auricles than usual. If the wave is premature, is out of place, it is called an extrasystole (Figs. 2, 3, 4, and 5). Premature auricular beats are usually followed by ventricular contractions. These often have (Fig. 4), but need not have (Fig. 5), the same form as the other complexes in the same individual. Many intermediate forms between the inverted and the normal wave occur. It is not yet possible to correlate each form with a special method of spread of the impulse. But changes in outline may occur (Fig. 6) without substantial

* We speak of the activity of the cardiac muscle in the usual terms, such as contraction, but it is understood that the accompanying electrical state is the matter of immediate concern.

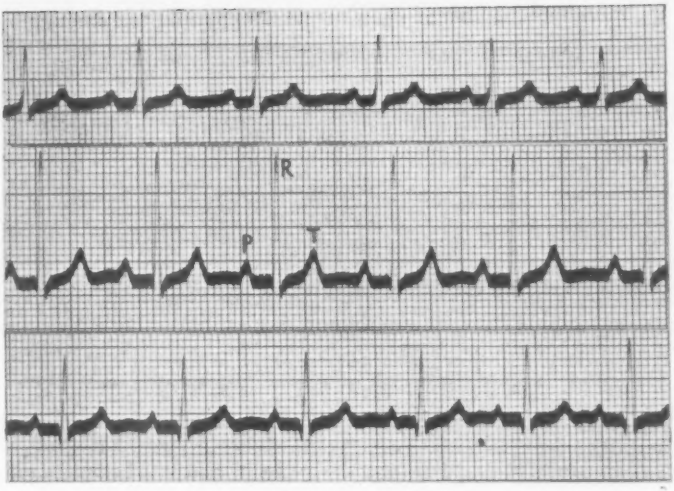

FIG. 1.—Normal electrocardiogram. Leads 1, 2, and 3.

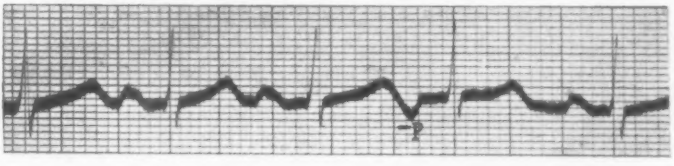

FIG. 2.—In the premature auricular beat (extrasystole) the auricular representative is inverted. Lead 2.

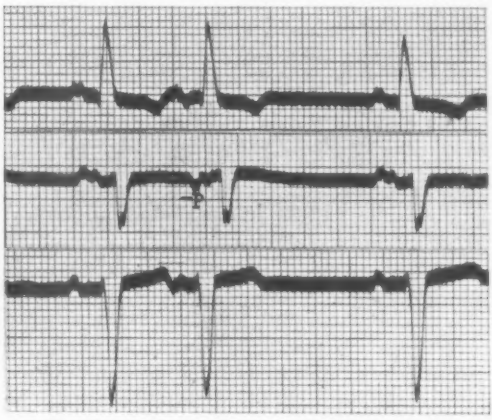

FIG. 3.—A single premature auricular beat taken from each of the three leads is shown. There is a tall *R* wave in L_1; the *R* wave is short and the *S* wave deep in L_2 and L_3. In L_2 the auricular representative is inverted. The ventricular complexes in the extrasystolic cycles are substantially like the usual beats.

alteration of rhythm.[5] When a given type of abnormal wave recurs in rapid succession, each followed by a ventricular beat, a paroxysm

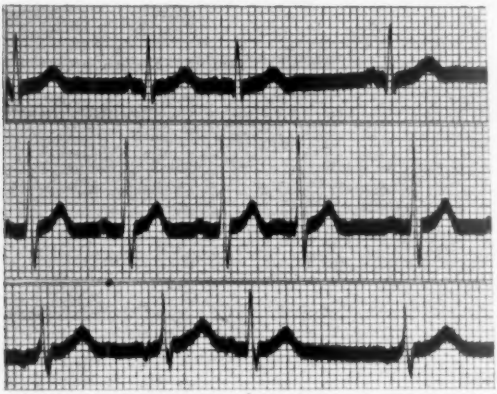

FIG. 4.—A single premature auricular beat taken from each of the three leads is shown. The extra-auricular representatives are like the usual beats. The ventricular complexes are also substantially like the usual beats.

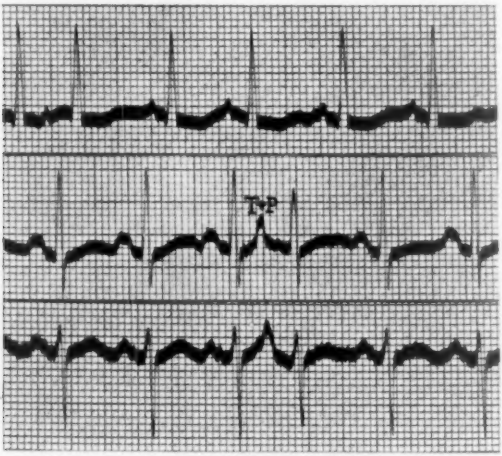

FIG. 5.—A single premature auricular beat taken from each of the three leads is shown. The extra-auricular representatives deform the preceding *T* waves. In L_2 and L_3 the *R* and *S* waves following the extra-auricular beats depart from the usual form.

of tachycardia (Fig. 7) exists.[6] [7] The clinical diagnosis depends in addition, of course, on the nature of the onset and offset. These

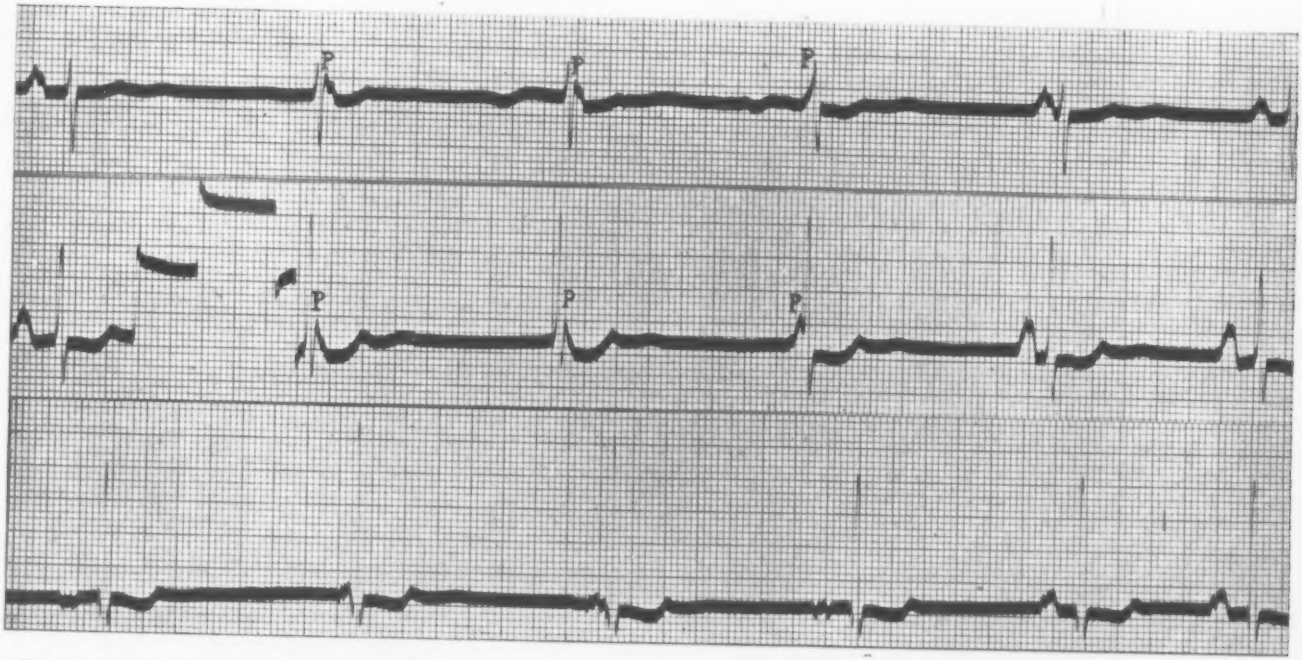

FIG. 6.—A form of nodal rhythm is shown. Leads 1, 2, and 3. Leads 1 and 2 deserve especial attention. They duplicate each other exactly in reproducing an entire phase of the irregularity. The relation of *P* to *R* waves vary in the same manner in each. In L_3 the *P*–*R* relation also varies; the sign of the *P* waves changes gradually from negative at the beginning to positive at the end of the curve. The *P*–*R* time at the beginning is a trifle longer than at the end of the curve. The patient from whom these curves were taken was under the influence of digitalis.

are the principal auricular changes. In the *ventricles* the passage of a normal wave of excitation is represented by two waves, *R* and

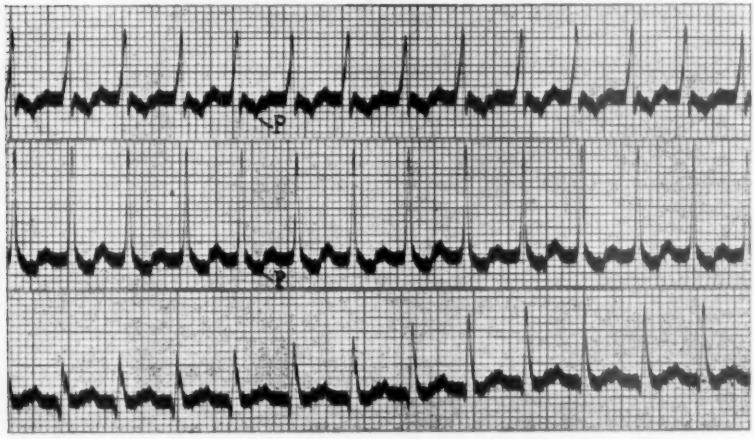

FIG. 7.—A portion is shown of a paroxysm of tachycardia of auricular origin. Leads 1, 2, and 3. The *P* waves in L_1 and L_2 are inverted.

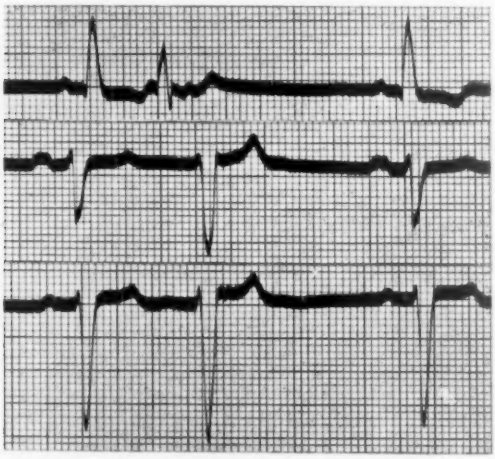

FIG. 8.—A single premature ventricular contraction is shown in each of the three leads. The extrasystole takes its origin in the wall of the left ventricle; there is a tall *R* wave in L_1 and a deep *S* wave in L_2 and L_3.

T (Fig. 1), the first a tall spike, the second a shorter but wider rounded wave, the two separated by a space. Systoles having an

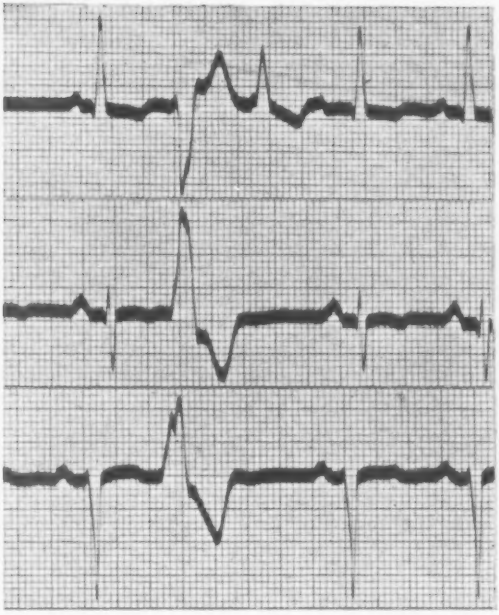

FIG. 9.—A single premature ventricular beat is shown in each of the three leads. In L_1 there is a deep *S* wave; in L_2 and L_3 the *R* wave is tall. In L_1 an added cycle resembling but differing somewhat from the other complexes follows the extrasystole. An explanation of its significance is not entered upon.

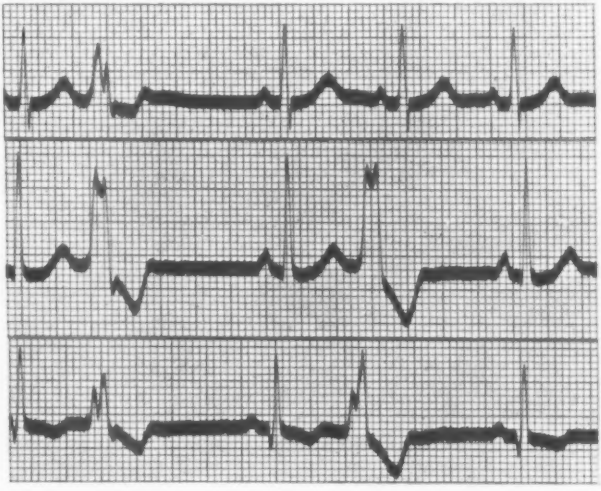

FIG. 10.—Premature contractions in each of the three leads are shown. Here the *R* waves are tall in all the extrasystoles.

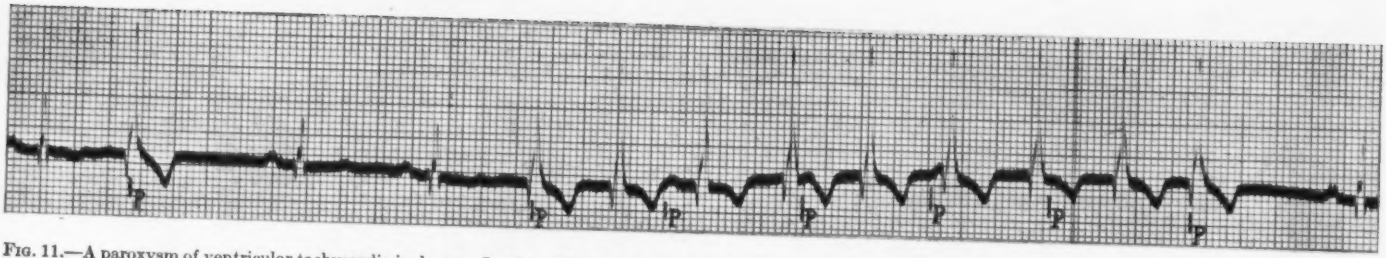

FIG. 11.—A paroxysm of ventricular tachycardia is shown. Lead 3. The paroxysm is composed of a succession of ectopic cycles like the second one reproduced. The paroxysm does not interfere with nor alter the regular recurrence of the auricular beats. Their incidence is indicated in the curve.

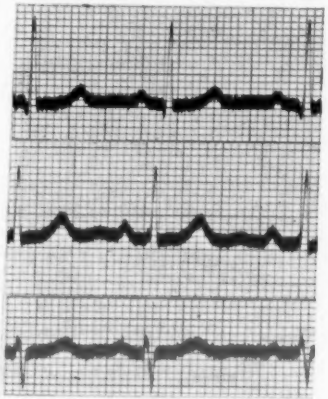

FIG. 12 A

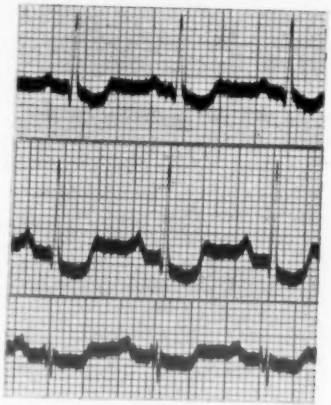

FIG. 12 B

FIG. 12.—*A*, leads 1, 2, and 3 taken from a patient not under the influence of digitalis. The *T* waves are upright. *B*, leads 1, 2, and 3 taken from the same patient under the influence of digitalis. The *T* waves are inverted in the three leads.

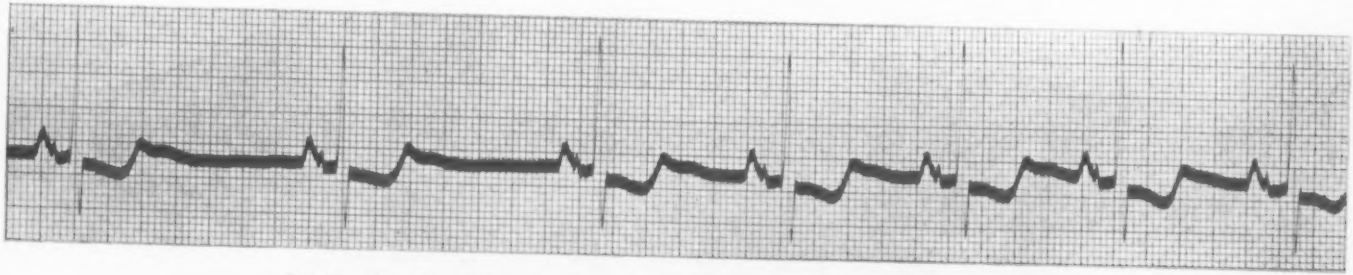

FIG. 13.—Sinus irregularity. Lead 2. The unequal spaces between succeeding cycles is shown.

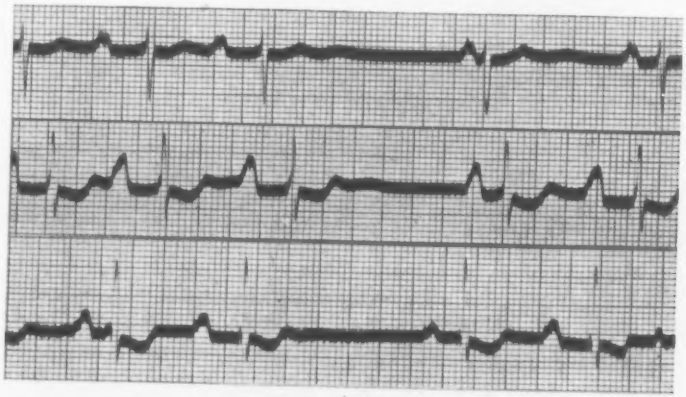

FIG. 14.—Sino-auricular block. Leads 1, 2, and 3. The long intervals between the *P* waves are equal almost to two of the other periods. A moderate sinus irregularity is also present.

abnormal origin[8] are detected by an alteration in the form of either of these waves, the altered form depending (1) on the site where the impulse starts, and (2) on the way the excitation wave travels. If it arises in the wall of the left ventricle it has one form (Fig. 8); if in the right ventricle, it has another (Figs. 9 and 10). When a beat of this nature is premature it is called an extrasystole. A rapid succession of such abnormal beats results in a paroxysm of tachycardia (Fig. 11).[9] Long paroxysms of ventricular origin are rare; usually they consist of a series of not more than twelve to thirty-six beats. In the paroxysm reproduced, two things are clear: (1) that the paroxysm consists of a succession of complexes, precisely like the single extrasystole which precedes it, and (2) the paroxysm leaves the rate and rhythm of the auricles undisturbed. Another alteration concerns the second ventricular wave, the *T*

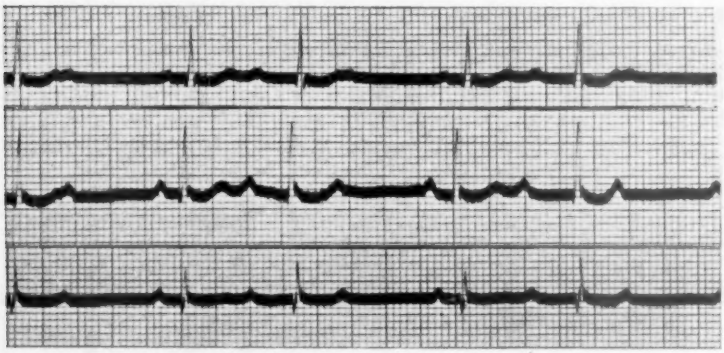

FIG. 15.—Incomplete auriculoventricular block. Leads 1, 2, and 3. The third auricular representative is blocked in each of the three strips. The patient is under the influence of digitalis. The *T* waves are inverted.

wave, and consists in an inversion (Figs. 12, *A* and *B*). It occurs under the influence of drugs of the digitalis group[10] and probably other agents.

The second of the factors underlying the normal electrocardiogram is the *sequence of contraction* of the chambers. Disturbance may occur in the regular succession of the activity of the pace-maker, as in sinus irregularity (Fig. 13), or disturbance of sequence may occur between the pace-maker (the sinus node) and the auricles, as in sino-auricular block (Fig. 14). Disturbance in sequence occurs also between the auricles and the ventricles (Figs. 15 and 16).[11] These are the familiar heart-blocks, and as rhythmic disturbances require no special description from the electrocardiographic point of view. But certain details of the ventricular mechanism in complete auriculoventricular dissociation have been brought to light

by this method. I cite a unique instance. In a case of complete heart-block the nature of the ventricular activity changed for a

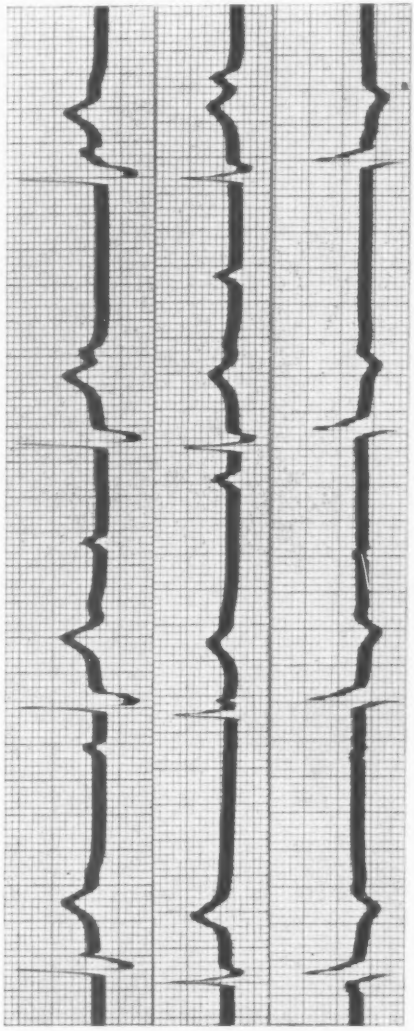

FIG. 16.—Complete auriculoventricular dissociation. Leads 1, 2, and 3. The varying relationship of the *P* waves to the ventricular complexes is shown in many phases.

time in an extraordinary manner. It was first of one form (Fig. 17, *A*) and then of another (Fig. 17, *B*). Electrocardiograms like this

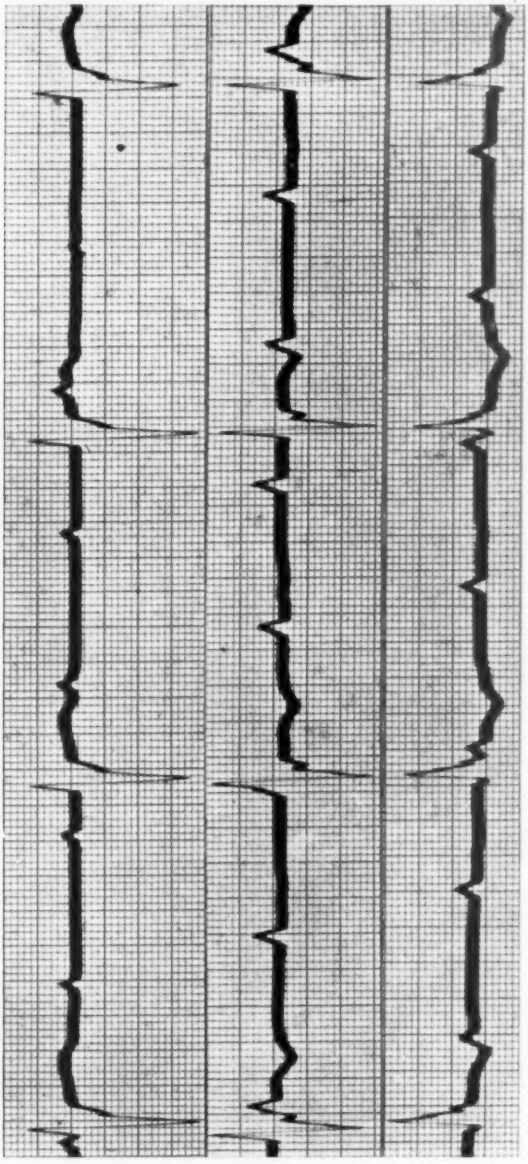

FIG. 17 A.—Complete auriculoventricular dissociation. Leads 1, 2, and 3. The ventricular impulses arise in wall of the right ventricle.

suggest that under circumstances not completely understood, one part and then another of the conduction system is rendered impassable. If the electrocardiogram returns to a former shape

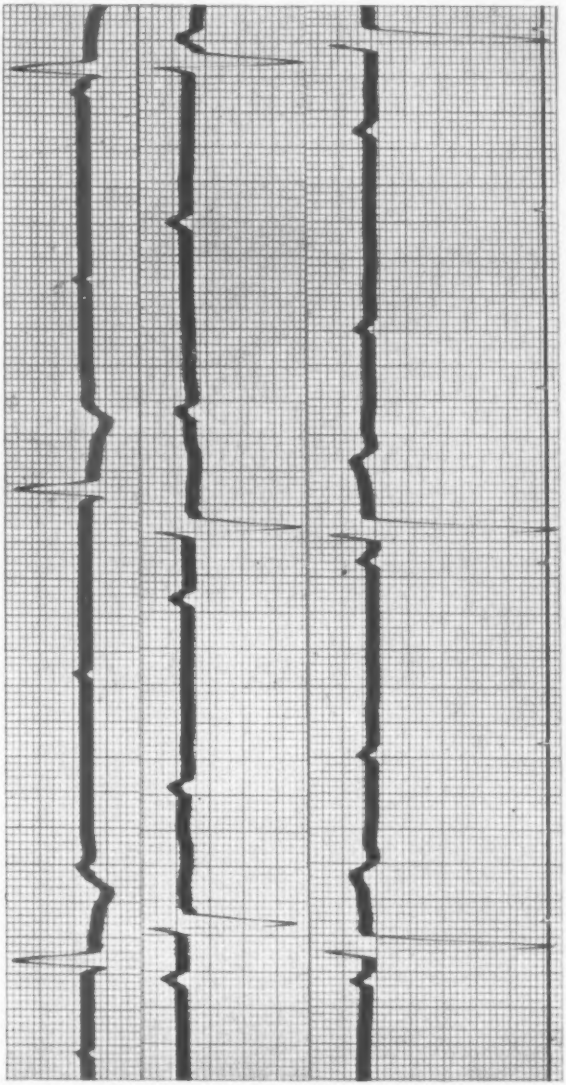

FIG. 17 B.—From the same patient. Leads 1, 2, and 3. The ventricular impulses arise in the wall of the left ventricle.

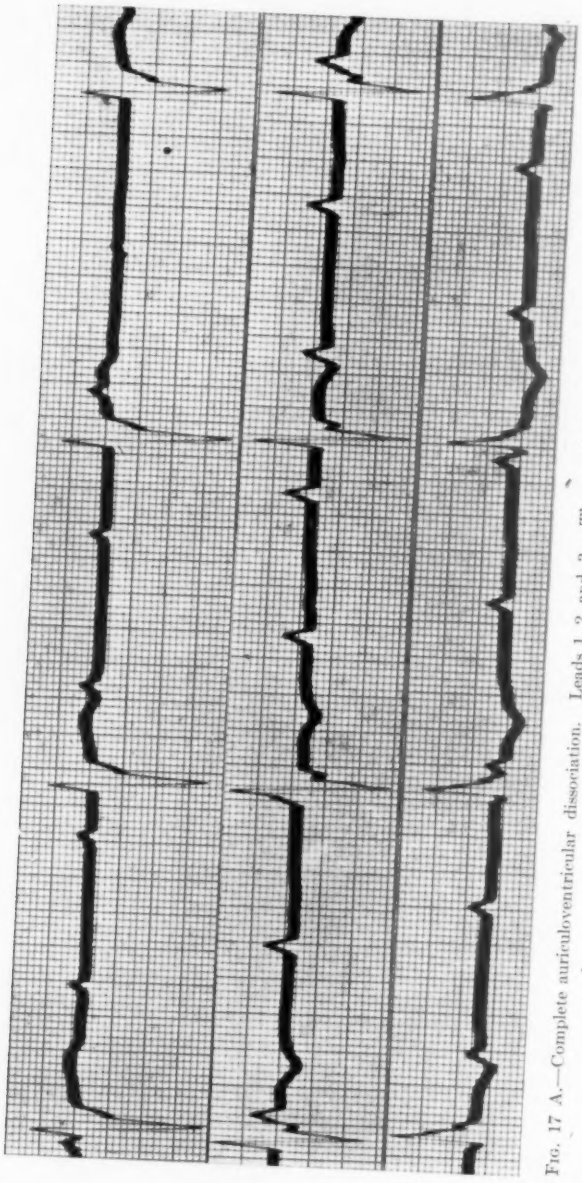

FIG. 17 A.—Complete auriculoventricular dissociation. Leads 1, 2, and 3. The ventricular impulses arise in wall of the right ventricle.

suggest that under circumstances not completely understood, one part and then another of the conduction system is rendered impassable. If the electrocardiogram returns to a former shape

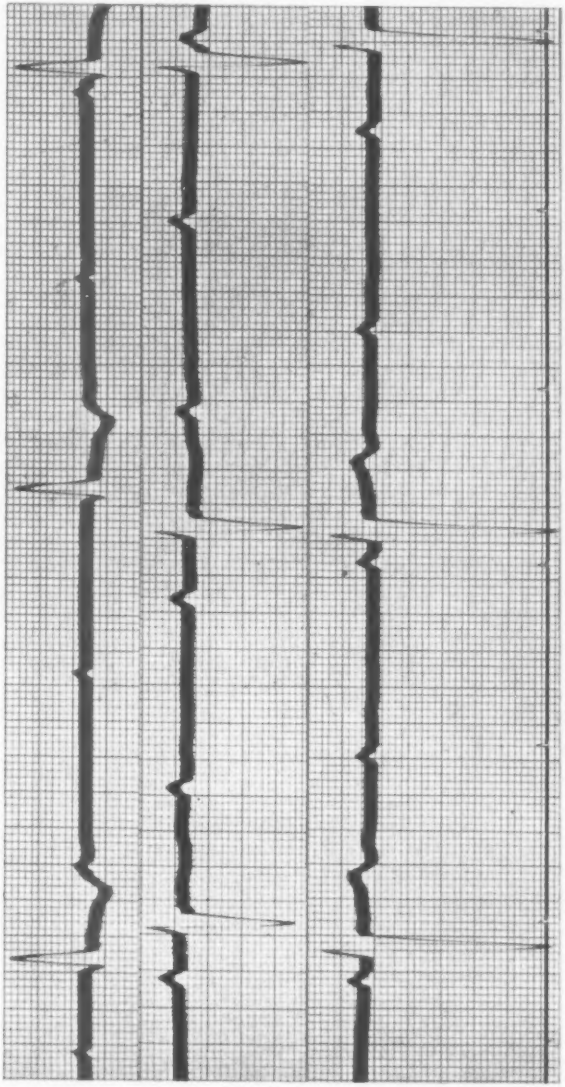

FIG. 17 B.—From the same patient. Leads 1, 2, and 3. The ventricular impulses arise in the wall of the left ventricle.

the cause must have been temporary, as in our case. If it does not, the cause is permanent and is probably an anatomical lesion. When the lesion involves one main branch of the *A–V* bundle, the left branch, for instance, a ventricular electrocardiogram results, having a form similar to that which occurs when the impulses spread from the right ventricle (Fig. 17, *A*). When, on the other hand, the right branch is injured the ventricular electrocardiogram is like that which occurs when the impulses spread from the left ventricle (Fig. 17, *B*). More recently electrocardiograms have been collected which, it is supposed, indicate that the main branches or small subdivisions

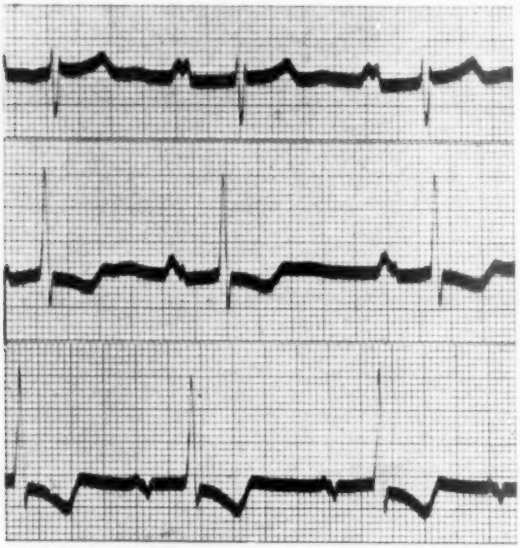

FIG. 18.—Long intervals between the *P* and *R* waves. Leads 1, 2, and 3. In L_1 in the first cycle the interval is 0.4 second. The patient was under the influence of digitalis.

of the bundle have received an injury. The recognition of these complicated forms has become commonplace since the introduction of the electrocardiographic method.[12]

Simpler defects in coördination between the auricles and ventricles also occur. The interval between the contraction of the two may be lengthened (Fig. 18). But a more interesting form is that in which the interval is shortened and may be changing. A common, not necessarily fixed, focus in the junctional tissue provides impulses which are conveyed upward to the auricles and downward to the ventricles. The impulses may reach both synchronously, the cases

of true nodal rhythm,[13] or may reach one before the other, or may reach them in an irregular fashion. In the latter event the length of the interval between auricular and ventricular contractions varies (Fig. 6). The mechanism then involves not only disturbance in sequence but also dislocation of impulse formation.

The third factor involved in the production of the normal electrocardiogram is the orderly *coördination of the muscular mass* composing each pair of cavities. The simplest form of disturbance of this mechanism occurs when the form of the auricular (*P*) wave is not rounded above but is split.[14] The two peaks are separated by a deep interval; sometimes the foot-points of the wave are widely separated (Fig. 18). This change has been associated with mitral stenosis, though it is impossible to say that it may not be found

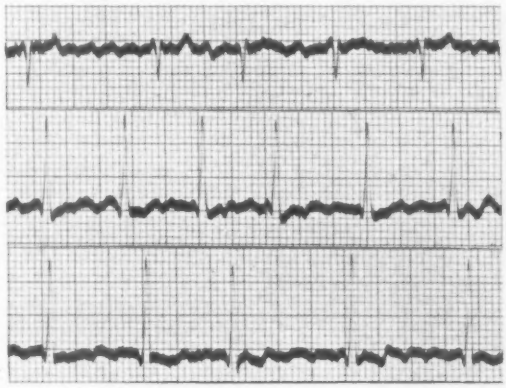

FIG. 19.—Fibrillation of the auricles. Leads 1, 2, and 3. Both fine and coarse waves are seen in the diastolic period.

under other circumstances,[15] and has been supposed to depend on a succession in time of the activity of the two auricles as the result of the mitral lesion.

The extreme example of incoördination in the auricles is seen in the completely irregular pulse which is now known to be due to fibrillation of the auricles (Fig. 19). Rothberger and Winterberg and Lewis[16] established this association by their electrocardiographic studies in 1909. The diagnosis of this rhythm is extremely important for it often furnishes the basis of satisfactory treatment not only in ordinary heart failure but in heart failure occurring in pneumonia and probably in other infections.

Flutter[17] (Fig. 20) is another condition which disturbs the orderly beating of the auricles. Its exact mechanism is still unknown, but its significance and treatment is like that of fibrillation. Its

detection is more difficult than that of fibrillation because occasionally the pulse remains quite regular. Like fibrillation, it may lead to heart failure on account of the resulting rapid rate of the ventricles. Making the diagnosis is important because treatment is often followed by the same satisfactory result as in fibrillation. The diagnosis may be made by other than the electrical method, but it is difficult and uncertain. It occurs spontaneously in pneumonia just as does fibrillation, and then gives rise to the same clinical state, and is controlled in the same way.

The failure of orderly coördination of the muscular mass within the ventricles is very much rarer than in the auricles. Two varieties have been described, and both can be detected only by the electro-

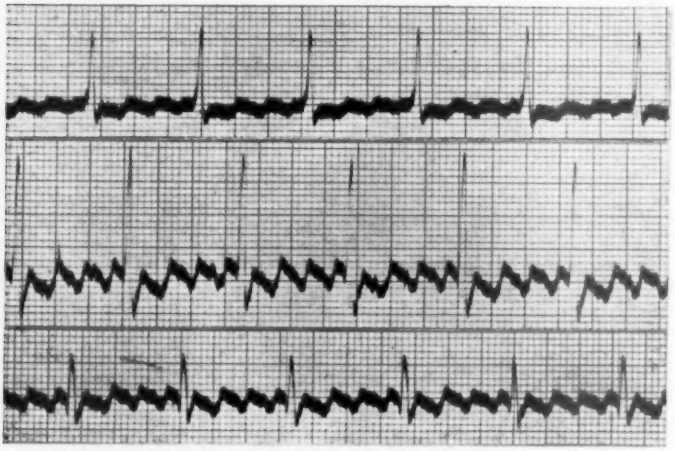

FIG. 20.—Flutter of the auricles. Leads 1, 2, and 3. Regularly recurring auricular representatives at the rate of 360 per minute are seen.

cardiogram. The first is associated with the sort of asynchronism between the two ventricles which, v. Leyden supposed, causes a split in the first sound.[12] Experiments and clinical observations have shown that this occurs in bundle branch lesions. I have already included the electrocardiographic signs of this condition, under disturbances in conduction, where it properly belongs. It is mentioned here because ventricular asynchronism which probably causes the split first sound results from the bundle defect.

A failure of orderly coördination resembling auricular fibrillation also occurs in the ventricles.[18] Its occurrence terminates life. That it is a method of death was suggested by Cushny, but the first genuine record of its occurrence was obtained by Robinson. There

is reason to believe that the onset of this rhythm is responsible for many cases of sudden death, especially in aortic insufficiency.

The fourth factor involved in the production of the normal electrocardiogram is the normal *volume and the usual arrangement* of the muscular mass in each pair of cavities. When these are disturbed, hypertrophy or destruction of the walls of the heart or dilatation of its cavities has taken place, or its position in the chest has altered. These abnormalities undoubtedly cause changes in the electrocardiogram (Fig. 21, *A* and *B*), but a correlation between the change in the heart and the change in the electrocardiogram has not yet been satisfactorily made. We are not in possession of the facts which are necessary before a solution of the problem is possible. Originally[19] electrocardiographic changes were believed to indicate corresponding anatomical alterations, but the electrocardiogram is no longer credited with giving that information. The difficulty has arisen because the electrocardiogram, although it sometimes shows the changes which the roentgenogram leads one to expect, does not *always* do so. The state of the matter is as follows: When the right side of the heart is enlarged, as in mitral stenosis, the ordinary three leads[20] yield ventricular curves, the spike of which is down in L_1 and up in L_3 (Fig. 21, *A*). When the left side of the heart is enlarged, as in aortic insufficiency, the ordinary three leads yield ventricular curves, the spike of which is up in L_1 and down in L_3, and often in L_2 (Fig. 21, *B*). In young children,[21] where the walls of the right side of the heart are relatively large, the curves resemble those of ordinary right-sided enlargement. In cases of congenital[22] cardiac defects, the curves of some resemble preponderant enlargement of one side, the curves of others of the other side. So far the situation is satisfactory, but there are many cases in which we find, for instance, apparently greater left-sided enlargement but fail to find evidence of this in the electrocardiogram. The curves may even be like those seen in right-sided preponderance. The difficulty probably lies in a failure to appreciate what information it is that the electrocardiographic method yields. It does not give an account of the distribution of the muscular mass, but an account of its electrical state. The same arrangement of the mass of the muscle should, and probably always does, give a curve of the same sort. If it does not, the arrangement of the muscle is probably not precisely that which it is believed to be. When it is remembered that knowledge of cardiac hypertrophy is limited, and that enlargement is usually taken to mean size in terms of the Roentgen-ray appearance, and not in terms of the complicated muscle layers and systems of the heart, it is clear why misapprehensions arise. The muscle layers of the heart are, in fact, not coextensive with a single cavity. They probably are strained and injured differently in different valve lesions, and the variations probably determine the shape, size, and constitution of the layers. The altered layers

balances and their electrical equivalents in view, may lead to a solution of the difficulty.

Alterations of the topography of the heart in the chest, due to displacements resulting from accumulations of fluid or air in the pleura or to dislocations resulting from adhesions, are responsbile for changes in the electrocardiogram as yet ill defined.[23] Transposition of the heart (dextrocardia)[24] is readily distinguished from displacements by this method. The electrocardiogram, lead for lead, is reversed; to be normal the usual first lead must be taken

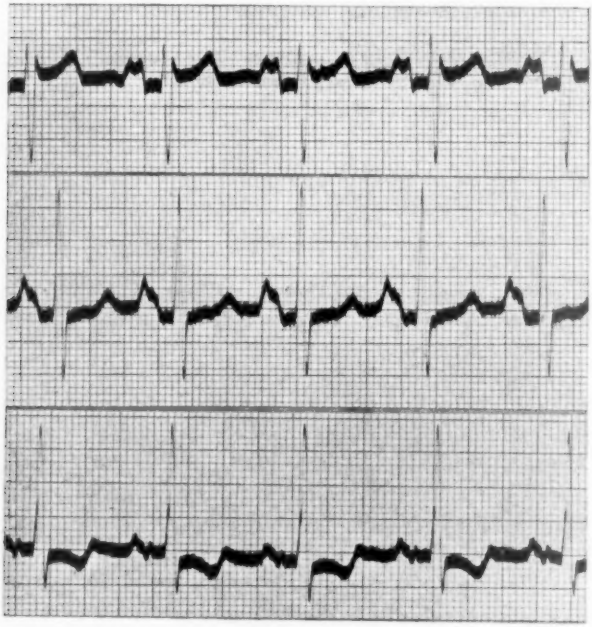

FIG. 21 A.—Curves from a case of mitral stenosis. Leads 1, 2, and 3. Split *P* waves are seen in the three leads; the foot-points of these waves are widely separated. There are short *R* and deep *S* waves in L_1; in L_2 and L_3 the *R* waves are tall and the *S* waves short.

from left arm to right arm; the second lead from left arm to right leg; the third lead from right arm to right leg.

The galvanometric method has also been used for recording heart sounds.[25] These have been registered by this method better than by any other so far used. The record is especially valuable when it is combined with the electrocardiogram by means of a two-string galvanometer or by a second instrument. It is unnecessary to describe the method or its results in detail. It has illuminated a

number of questions which have been of interest in auscultation, especially in relation to the auricular systolic murmur in mitral stenosis and its fate when the auricles fibrillate. It has illuminated these questions, but it has contributed nothing new in principle.

It was anticipated, unwisely, that the method would also contribute toward an estimation of the work power of the heart, of its functional efficiency. From this point of view, electrocardiography has been disappointing. But the development of electrical potential, of which the galvanometric method gives information, is only one of the functions of muscular activity. An account of a single function may, naturally, on occasion, be an index of the entire activity

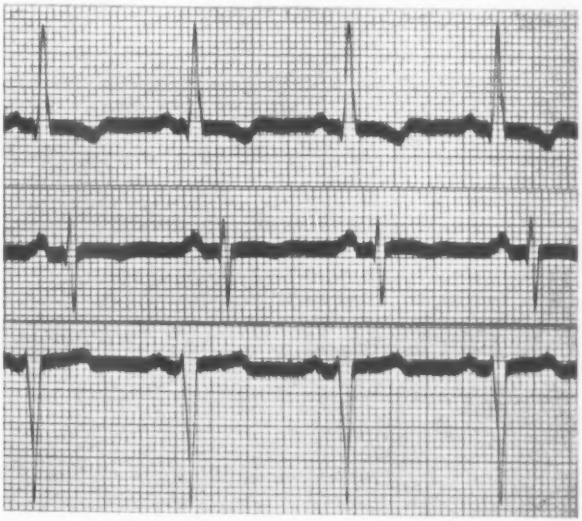

FIG. 21 B.—Curves from a case of cardiosclerosis. In L_1 there are tall *R* waves. In L_2 the *R* waves are shorter and moderate-sized *S* waves appear. In L_3 there are small *R* waves but deep *S* waves.

of a tissue. But the estimation of electrical discharge has so far not been such an indicator. More detailed observation and closer analysis may indicate capacities which are not yet appreciated.

The description of the normal electrocardiogram and its principal alterations observed in clinical medicine is thus concluded. When the subjects reviewed are grouped under the topics familiar in cardiac pathology, they are seen to be included under headings of irregularities in mechanism, hypertrophy and dilatation, and malone may easily believe, alter the electrical values with which we are immediately concerned. Investigations, keeping these muscular

positions and displacements. To these subjects the method has yielded added information. We are now able to think of mechanism in greater detail, as in fibrillation and flutter. Asynchronism of the chambers can now probably be demonstrated. The exact origin and method of the passage of impulses has become a matter of importance, as in paroxysms of tachycardia. In the detailed study of individual waves, notice is taken of alterations which are of consequence in treatment, as in the *T* wave changes arising under the influence of digitalis. In recognizing the value of small alterations of this kind it has become clear that each change in the electrocardiogram may be a change of significance, and may not be dismissed as of no importance.

I have in this review collected data which may be grouped under four headings: (1) those relating to arrhythmias, the mechanism of which has been brought to light by the electrical method; (2) certain facts relating to the *passage of impulses* through the auricles and ventricles, a knowledge of which is being increasingly valuable in diagnosis and in treatment; and (3) certain considerations in regard to the *size* of the heart and (4) its position in the chest.

REFERENCES.

1. Einthoven, W. Le Télécardiogramme, Arch. Internat. de Physiol., 1906, iv, 132; Weiteres über das Elektrokardiogramm, Arch. f. d. gesam. Physiol., 1908, cxxii, 547.

2. The figures are given by Lewis, Thos., and Gilder, M. D. D. Philos. Trans., Roy. Soc., London, 1912, Series B, ccii, 351–376; and by Kahn, R. H., Ergeb. d. Physiol., 1914, xiv, 35–36.

3. Kraus, F., und Nicolai, G. F. Ueber das Elektrokardiogramm unter normalen und Pathologischen Verhältnissen, Berlin. klin. Wchnschr., 1907, xliv, 811.

4. James, W. B., and Williams, H. B. The Electrocardiogram in Clinical Medicine, Amer. Jour. Med. Sci., 1910, cxl, 651–2.

5. Cohn, A. E., and Fraser, F. R. Certain Effects of Digitalis on the Heart, XVII Internat. Cong. Med., London, 1913, Section vi, 257.

6. Lewis, Thos. Paroxysmal Tachycardia, The Result of Ectopic Impulse Formation, Heart, 1909–10, i, 262–282.

7. The Explanation of Paroxysms of Tachycardia given by Mines, G. R., Trans. Roy. Soc. of Canada, 1914, viii, 3d series, 48 is not at variance with this statement.

8. Einthoven, W. Weiteres über das Elektrokardiogramm, loc. cit., p. 578; Le Télécardiogramme, loc. cit., p. 150.

9. Hoffmann, A. Die Arhythmie des Herzens in Elektrokardiogramm, München. med. Wchnschr., 1909, p. 2262.

Lewis, Thos. The Mechanism of the Heart Beat, London, 1911, p. 168.

10. Cohn, A. E., Fraser, F. R., and Jamieson, R. A. The Influence of Digitalis on the *T* Wave of the Human Electrocardiogram, Jour. Exper. Med., 1915, xxii, 593–604.

11. Einthoven, W. Le Télécardiogramme, loc. cit., p. 159; Weiteres über das Elektrokardiogramm, loc. cit., p. 582.

12. Eppinger, H., and Rothberger, J. Ueber die Folgen der Durchschneidung der Tawaraschen Schenkel des Reitzleitungssystem, Ztschr. f. klin. Med., 1910, lxx, 1–20.

Eppinger, H., and Stoerk, O. Zur Klinik des Elektrokardiogramms, Ztschr. f. klin. Med., 1910, lxxi, 157–164.

Lewis, Thos. Observations upon Disorders of the Heart Beat, Heart, 1912, iii, 293 ff.

13. Williams, H. B., and James, H. Reversal of the Cardiac Mechanism, Heart, 1913–14, v, 109–118.

14. Einthoven, W. Le Télécardiogramme, loc. cit., p. 148.
15. Lewis, Thos., and Gilder, M. D. D. Loc. cit., pp. 362–363.
16. Rothberger, J., and Winterberg, H. Vorhofflimmern und Arhythmia Perpetua, Wien. klin. Wchnschr., 1909, xxii, 839–844.
Rothberger, J., and Winterberg, H. Ueber das Elektrokardiogramm bei Flimmern der Vorhöfe, Arch. f. d. gesam. Physiol., 1910, cxxxi, 387–408.
Lewis, Thos. Auricular Fibrillation, a Common Clinical Condition, British Med. Jour., 1909, p. 1528.
17. Hertz, A. F., and Goodhart, G. W. The Speed Limit of the Human Heart, Quart. Jour. Med., 1909, ii, 213–218.
Ribl, J. Hochgradige Vorhoftachysystolien mit Ueberleitungstörungen und elective Vaguswirkung, Ztschr. f. Exp. Pathol. u. Therap., 1911, ix, 277.
Cohn, A. E. Auricular Tachycardia with a Consideration of certain Differences between the Two Vagi, Jour. Exper. Med., 1912, xv, 49–62.
Jolly, W. A., and Ritchie, W. T. Auricular Flutter and Fibrillation, Heart, 1910–11, ii, 177–221.
18. Hoffmann, A. Demonstrationen zur Lehre von der Form des Elektrokardiogramms, Verhandl. d. Kong. f. Innere Med., Wiesbaden, 1911, xxviii, 429; Fibrillation of the Ventricles at the End of an Attack of Paroxysmal Tachycardia in Man, Heart, 1912, iii, 213.
Robinson, G. C. A Study with the Electrocardiograph of the Mode of Death of the Human Heart, Jour. Exper. Med., 1912, xvi, Figs. 8 and 19.
Halsey, R. H. A Case of Ventricular Fibrillation, Heart, 1915, vi, 67–76.
19. Einthoven, W. Le Télécardiogramme, loc. cit., p. 147; Weiteres über das Elektrokardiogramm, loc. cit., p. 568.
20. L_1 right arm to left arm. L_2 right arm to left leg. L_3 left arm to left leg.
21. Funaro and Nicolai, G. F. Das Elektrocardiogramm des Säuglings, Zentralbl. f. Physiol., 1908, xxii, 58.
22. Einthoven, W. Le Télécardiogramme, loc. cit., Fig. 24; Weiteres über das Elektrokardiogramm, Fig. 35.
Wandel. Untersuchungen über die Aktionsströme des Herzens, München. med. Wchnschr., 1908, xlv, 2356.
Steriopulo, S. Das Elektrokardiogramm bei Herzfehlern, Ztschr. f. Exper. Pathol. u. Therap., 1909, vii, 467–493.
Nicolai, G. F. Das Elektrokardiogramm bei Dextrokardie und anderen Lageveränderungen des Herzens, Berl. klin. Wchnschr., 1911, p. 54.
23. Grau, H. Ueber den Einfluss der Herzlage auf die Form des Elektrocardiogramms, Ztschr. f. klin. Med., 1909, lxix, 281–290.
24. Nicolai, G. F. Das Elektrokardiogramm bei Dextrokardie und anderen Lageveränderungen des Herzens, loc. cit., pp. 51–55.
Samojloff, A. Vorzüge der mehrfachen Ableitung der Herzströme bei Elektrokardiogramm aufnahmen, illustriert an zwei Beispielen, Arch. f. d. gesam. Physiol., 1913, cliii, 196–218.
25. Einthoven, W. Die Registrierung der menschlichen Herztönen mittels des Saitengalvanometers, Arch. f. d. gesam. Physiol., 1907, cxvii, 461–472; Ein drittrer Herzton, Ibid., 1907, cxx, 31–43.

THE OCCURRENCE OF MILIARY TUBERCULOSIS OF THE LIVER IN THE COURSE OF PULMONARY TUBERCULOSIS.

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I have recently made a study of a series of autopsy reports, giving the records of the findings in 131 cases of pulmonary tuberculosis. The object of the investigation was primarily to observe

certain points of interest regarding the tuberculous involvement of the liver and other abdominal organs. Chronic tuberculosis of the liver with large masses of tuberculous material occurs commonly in certain animals, and a few cases have been reported in human beings; but this condition is so rare in man that it is considered as a pathological curiosity, and need not be considered in this discussion.

There is no dearth of literature regarding the frequency of occurrence of tuberculosis in the liver. Ullom's[1] article covers the subject with thoroughness, and Rolleston[2] gives an up-to-date *résumé* of the subject, with references. White[3] has discussed thoroughly the occurrence of miliary tubercles in the liver in the cases studied by him.

It is generally recognized that the liver is involved by miliary tuberculosis in a large percentage of fatal cases, Rolleston putting the figures at about 50 per cent. and White as high as 80 per cent. The opinion of most authors leads us to believe that an involvement of the liver by a tuberculous process is a matter of little importance, and that for some reason it is likely to occur just before death. This view, which seems to be expressed by most writers on the subject, is far from satisfying. I am not aware that I have ever seen an explanation as to why a widespread miliary tuberculosis should occur just before death in the liver, though it does not develop in other organs which ordinarily do not show tuberculous formation, as the pancreas. Tubercle bacilli have been demonstrated in the bile, and they must be passed through the liver, more or less constantly, or deposited in its spaces throughout the course of chronic pulmonary disease.

Much work has been done on the question of the effect of tubercle bacillus in the production of fibrosis of the liver, and it seems probable, as held by Stoerck and others,[4] and as confirmed by the observance of Lavenson and Karsner,[5] that a gradual infection of the liver can take place with a disappearance of the tubercles as formed, resulting in a periportal fibrosis. This does not, however, in any way explain the facts found at autopsy, where we find, as a rule, a wide distribution of young miliary tubercles of approximately the same age, seeming to indicate an overwhelming infection. It would seem necessary to consider whether this condition can exist without serious result.

The following points have been observed: In the liver, miliary tuberculosis is usually demonstrable microscopically, the tubercles fairly young in state of development and occurring in the peri-

[1] Amer. Jour. Med. Sci., 1909, cxxxvii, 694.
[2] Diseases of the Liver, Gall-bladder, and Bile Ducts, London, 1912, 2d ed.
[3] Fifth Annual Report of the Henry Phipps Institute, Philadelphia, 1909.
[4] Wien. klin. Wchnschr., 1907, xx, 847.
[5] Univ. Penna. Med. Bull., Philadelphia, 1909, xxii, 167.

lobular spaces, about the portal vessels, with the greatest frequency. The tubercles are usually not demonstrable grossly, unless there is present a miliary tuberculous peritonitis with invasion of the liver capsule. There may be perilobular fibrosis, and there is likely to be considerable degree of fatty change. In the kidney, tubercles are seen grossly in a much larger proportion of cases than they are found in histological examination, as noted by White. Occasionally old lesions may be seen on the kidney, either caseous or fibrous, or perhaps a single young tubercle; but in histological sections cut for the purpose of examining the kidney structure a tubercle has very seldom been encountered. In the spleen they are found much more frequently than in the kidney, and in the spleen they are older, as a rule, than in the liver. It is impossible to say just how old these gray tubercles found in the liver are, but in working with rabbits, Evans, Bowman, and Winternitz,[6] in studying the pathogenesis of the tubercle of the giant cell in experimental tuberculosis, find a beginning of tubercle formation within six hours after injection of the bacilli into the mesenteric vein and well-developed tubercles within forty-eight hours.

The 131 cases studied in this series have been divided into four classes:

Class A.—Cases apparently dying of pulmonary tuberculosis, showing miliary tubercles of the liver; these are, as nearly as can be determined from the autopsy records, and in some cases from a study of clinical histories, cases which may properly be said to have died from pulmonary involvement, that is, not dying from such intercurrent causes, as violent hemorrhage, advanced amyloid disease, severe pneumonia with acute consolidation of the lung tissue, etc. These cases all show miliary tuberculosis microscopically, but in comparatively few it is demonstrable grossly.

Class C.—These are cases which would be diagnosed at autopsy as more or less generalized miliary tuberculosis, usually showing miliary tubercles of the peritoneum, and possibly of the meninges, spleen, liver, and perhaps of the bladder; but here particularly are found cases of omental and peritoneal involvement, in which it seems that a spread to the liver by way of the lymphatics of the capsule has taken place. A marked point of difference between this class and the first is that in these cases the miliary tuberculosis present in the liver is nearly always recognized grossly. These cases all show miliary tuberculosis of the liver.

Class D.—These are cases of advanced tuberculosis where death is due to some intercurrent event, as acute cardiac dilatation, acute pneumonia, etc. (In this is included tuberculous pneumonia, where consolidation of both lungs is almost complete.) These cases do not show miliary tubercles of the liver.

[6] Jour. Exper. Med., 1914, xix, 283.

Class X.—These are cases apparently dying from pulmonary tuberculosis in which tubercles were not reported on histological examination. It is difficult to classify these cases, but we feel that on the average it has been done fairly. There are probably several cases under "C" which really belong in classification "A," and there are some cases under "X" which should probably be placed in "D." Mention should be made of the question of tuberculous pneumonia, in which there was consolidation of nearly all of the lung tissue. This was considered as the cause of death under classification "D," but considerable degrees of consolidation, if of a tuberculous nature, have been classified under "A" or "X."

A summary of the results show: "A," 49 cases; "D," 29 cases; "C," 33 cases; and "X," 20 cases. Eighty-two cases, or about 63 per cent., therefore, showed miliary tuberculosis of the liver. What appears more significant, however, is that the 29 cases classed under "D," all of which showed active pulmonary tuberculosis, but with death from independent causes, or as the result of acute complications, none showed miliary tubrcles in the liver. This is the point, suggested by this compilation, which demands explanation. It would seem that the liver can take care of a gradual tuberculous infection, but that a sudden and overpowering infection of the portal circulation overwhelms the liver resistance, and there ensues a sudden development of disseminated miliary tuberculosis. It is reasonable to assume that this must have a profound constitutional effect, and in the case of an individual weakened by a long course of chronic disease, this might readily lead to a fatal result.

Infection of the portal circulation may take place either from the intestines, which are nearly always infected, or from the spleen. Mechanically the ideal source of a massive infection of the portal stream lies in the spleen, in which the tubercles are apt to be comparatively large, of varying age, and lying in close relation to large blood spaces leading directly into the portal stream.

While it is unsafe to draw parallels between human tuberculosis and experimental tuberculosis in animals, the work of Lewis and Margot,[7] showing an apparent increase in resistance to tuberculosis after the removal of the spleen in mice, suggests that the spleen may be a source of infection to the liver.

There is need for considerable rseearch work to determine the part played by the liver in fatal tuberculosis. If this series of cases is representative, and if, as a rule, these findings are present, viz., that in cases dying from pulmonary tuberculosis there is usually present a wide-spread miliary tuberculosis of apparently recent onset, and in cases dying from other causes this is not found, we must consider two possible explanations for these findings: (1) that in cases dying from tuberculosis the liver tissue, for some

[7] Jour. Exper. Med., 1915, xxi, 84.

reason, loses its resistance to the tubercle bacilli shortly before death, and at the same time is freely infected through the portal circulation, or (2) that in these cases the liver is able to cope with a gradual infection, but a sudden and overwhelming flooding of the portal stream with the tubercle bacilli overpowers the hepatic resistance, allowing tubercles to develop, and that the reaction in the liver has a severe constitutional effect, often determining the fatal issue.

In view of the fact that we do not find miliary tuberculosis developing in the liver in cases of active tuberculosis dying from independent causes, and that other tissues of the body are not rendered susceptible to tubercle development with the liver as death approaches, the latter explanation seems more reasonable than the former. I think it probable that such infection takes place by way of the portal circulation, and probably very frequently from the spleen. This view accords, in a measure, with the observation that experimentally the development of fatal tuberculosis is delayed by the removal of the spleen.

I desire to express my sincere appreciation to Prof. Allen J. Smith, of the University of Pennsylvania, both for valuable suggestions and for placing at my disposal the autopsy records of the Pathological Department of the University, and to Dr. H. R. M. Landis, director of the Phipps Institute of the University of Pennsylvania, for allowing me to refer to the autopsy records of that institute.

Cases apparently dying of pulmonary tuberculosis and showing miliary tuberculosis of the liver on microscopic examination:

CLASS A.

1900—	26	
	34	
	54	
	35	Tuberculous pneumonia.
1901—	64	
	86	
	91	
	107	
1902		
(?) —	5	Pneumonia and disseminated tuberculosis.
	73	
1903		
(?) —	12	Locomotor ataxia.
1904—	15	
	24	
	26	
	35	
	37	
	66	Miliary tuberculosis of lungs and pneumonia.
	79	
	98	Tuberculous pneumonia.
	120	
	132	
	157	
	158	
	202	Tuberculous pneumonia.
	24	

CLASS A (Continued).

Balance forward—	24	
1905—	1	Acute parenchymatous nephritis.
	6	Tuberculous pneumonia; endocarditis (mitral).
	27	Hemiplegia; amyloid; tuberculous pneumonia.
	90	
	108	
	112	
	123	
	138	
	157	
	4659	
	4587	
	4522	
	4500	
	4462	
	4460	
	4380	
	4306	
	4281	
	4282	
	4272	
	4248	Recent hemorrhage, eight ounces.
	4232	Chronic advanced pulmonary tuberculosis. At right base there is pneumonic consolidation, but no microscopic examination of lungs (pneumonia ?).
	4198	Also amyloid liver and spleen.
	4155	
	4148	Hypernephroma of kidney, but thoroughly encapsulated and no metastasis.

49 cases.

Cases of more or less generalized tuberculosis, usually showing involvement of peritoneum and omentum, or of general miliary tuberculosis. All show miliary tuberculosis of the liver:

CLASS C.

1900—	61		Balance forward—	18	
1901—	28		1904—	142	
	114			170	
	124			174	
	214			190	
	220			196	
1902—	6			213	
	75		1905—	3	
	191	Peritonitis.		9	
	212			46	
	245			47	
1903—	152			49	
1904—	9			69	
	17			110	
	63			126	
	110			140	
	117				
	131			33	cases.

18

Cases of pulmonary tuberculosis dying from other causes and not showing miliary tuberculosis of the liver on microscopic examination:

CLASS D.

1900—	55	Advanced amyloid, aortic, and mitral valvulitis (veg.), hydropericardium; tuberculosis (primary); epididymitis and scrotum.
1901—	48	Ulcer of stomach and hemorrhage into pancreas; chronic and
	154	acute miliary pulmonary tuberculosis, and abscess; perforation of esophagus.
1902—	15	Rheumatoid arthritis; advanced amylosis of liver, kidney, spleen, etc.
	16	Diabetes; pulmonary gangrene.
	30	Chronic pulmonary tuberculosis; acute bronchopneumonia; acute infiltrated liver; portal space infiltrated.
(?)	65	Cancer of bowels; edema of lungs (?); tuberculous ulcer; mesenteric nodes.
	182	Pyelonephritic tuberculosis; subacute fibroid pleurisy; edema of brain (?); tuberculosis (?).
	202	Psoas abscess; miliary tuberculosis of lungs; chronic mitral valvulitis; pulmonary hemorrhage.
1903—	42	Pulmonary tuberculosis; pneumonia.
	154	Acute dilatation of heart following operation of glands of neck.
1904—	6	Nephritis; dropsy; operation for decapsulation; pulmonary fibrosis and fibroid tubercles.
	18	Operation on tuberculous shoulder-joint followed by miliary tuberculosis of lungs and bronchopneumonia.
	86	Tuberculous meningitis; internal hydrocephalus.
	105	Tuberculous pneumonia; complete of right and almost complete consolidation of left.
	147	Cardiac hypertrophy; valvulitis; bronchopneumonia; tuberculosis.
	171	Hypertrophy and dilatation of heart; hydropericardium; tuberculous pneumonia.
	184	Fibroid tuberculosis of left lung; miliary tuberculosis of right lung; edema and congestion; mitral and aortic disease; nephritis.
	187	Myocarditis; acute endocarditis; chronic bronchitis; both apices.
(X) ?	216	Fibroid and fatty heart; calcified aortic leaflets.
	233	Septicemia; peritonitis.
1905—	15	Amyloid spleen, liver, kidney; carcinoma of uterus and kidney.
(X)	26	Tuberculous pneumonia; tuberculous spleen; pyothorax. (One liter of pus.)
	44	Tuberculous vertebræ; amyloid liver, kidney, and spleen—advanced.
	64	Pulmonary tuberculosis; tuberculous pneumonia; pulmonary congestion; brown atrophy of heart; chronic nephritis. Aged eighty years; tuberculous spleen.
	106	Miliary tuberculosis of lungs and peritoneum; acute nephritis; death following operation on ileum and sigmoid; no tubercles in spleen.
	159	Empyema; collapse of lung; cloudy swelling of kidney; tuberculous kidney and spleen.
	4364	Tuberculous pneumonia, acute; both lungs practically solid.
	4243	Acute pneumonia; sudden death.

29 cases.

Cases in which death seems to be a result of pulmonary tuberculosis and which do not show miliary tuberculosis of the liver on microscopic examination:

CLASS X.

1901—	5	Apparently pulmonary tuberculosis, but also edema of brain and gelatinous pneumonia. (Microscopic.)
	36	Fatty and parenchymatous degeneration of liver.
	103	
	117	
1904—	42	Tuberculous meningitis (miliary tubercles).
	100	Acute parenchymatous nephritis.
	116	Cardiac fibrosis; bronchopneumonia.
	130	Myocarditis, fibrosis, and fatty infiltration.
	176	Brown atrophy of heart; fibroid lung (left); ulcerative tuberculosis of right lung.
1905—	13	Chronic myocarditis; chronic diffuse nephritis.
	33	Tuberculous pneumonia.
	36	Nephritis; myocarditis; pulmonary edema and congestion.
	135	Tuberculous pneumonia of right lung (1120 grams), entirely solid; left, slight.
	4415	Pathological diagnosis written by pathologist personally states miliary tuberculosis of liver; detailed report does not state.
	4345	Amyloid liver, spleen, kidney, intestines, etc.
	4287	Few tubercles grossly. Not reported microscopically.
	4224	Typical course of chronic pulmonary tuberculosis; tuberculosis in liver reported grossly, but not mentioned in histological report.
	4197	Same as 4224. Reported grossly.
	4221	
	4316	

20 cases.

ANALYSIS OF ONE HUNDRED AND THIRTY-FOUR CASES OF BACTERIEMIA.

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Excepting the statements in the monographs of Canon[1] and Lenhartz,[2] and the brief summaries of Soper,[3] Jochmann,[4] Burnham,[5] Bertelsmann,[6] and a few others, it is difficult to find statistics of volume sufficient to have value in judging the prognosis of bacteriemias. Bertelsmann summarizes 49 cases of bacteriemia, Canon 98, Lenhartz 90, Soper 29, Burnham 111.

In the study here presented we have tabulated 134 cases of

[1] Die Bacteriologie des Blutes bei Infektionskrankheiten, Jena, 1905.

[2] Die septischen Erkrankungen, Nothnagel, Spez. Path. and Therap., Wein., 1903, iii, 4, 1, 197.

[3] A Series of Cases of Staphylococcus aureus sepsis, Proc. New York Path. Soc., 1912, N. S., xii, 223–225.

[4] Handbuch der inn. Med., 1911, i, 655.

[5] Vaccine and Serum Therapy in Septicemia, Ann. of Surg., 1914, lix, 5, 653.

[6] Die Allgemein Infection bei chirurgischen Infektionskrankheiten, Deutsch. Ztschr. f. Chir., 1904, lxxii, 209.

bacteriemia observed in the various services of the Roosevelt Hospital during the past five years. There are recorded in the briefest possible way. The hospital case number, the age of the patient, the diagnosis, the maximum temperature, the leukocyte count and polynuclear percentage, the bacteriological blood findings, the treatment, and the result are given.

STATEMENT OF LABORATORY METHODS. So far as possible the number of colonies which developed in a poured agar plate from approximately 1 c.c. of blood is entered under the caption blood culture. The result is otherwise indicated as No growth in solid media, Fluid media only used, or Number not noted. From this one gains some knowledge of the grade of bacteriemia represented by the viable organisms present per c.c. in the circulating blood at the time of culture.

We have found the following to be a satisfactory method for routine use; 2 or 3 c.c. of blood are delivered into a French square bottle of a half-pint capacity containing 75 c.c. of infusion broth (acid +0.6); 5 to 6 c.c. into the same type of bottle of one pint capacity containing 200 c.c. of broth of like character, and 1 to 2 c.c. are poured into a Petri plate with 6 to 8 c.c. of agar (acid +0.6) melted and cooked to 42° to 45° C. In many instances additional media were used, including glucose agar and glucose broth with and without the addition of serum or ascitic fluid, glycerin agar, and broth media; also media of varying acidity. At times a portion of blood for examination was treated by one or more of the following means before cultivation: (1) defibrination, (2) allowing coagulation to take place and pouring off the serum, (3) decalcification with potassium oxalate or sodium citrate, (4) laking with distilled water, (5) collection into a flask containing crystal salt enough to make about a 5 per cent. solution of salt in the blood added.

It is unnecessary to make any remarks on the bacteriological classification of the cases other than those of the Streptococcus group. Under Streptococcus hemolyticus are included 26 cases in which the organism in question was a Gram-positive streptococcus whose colonies in blood agar presented a typical zone of clear hemolysis; and cases Nos. 24, 28, and 104 (all pelvic sepsis); case No. 47 an atypical streptococcus producing diffuse hemolysis and recorded as being dissolved by bile; case No. 14 a conglomerate streptococcus with slight hemolytic activity.

Under Streptococcus viridans are included as follows:

I. A Gram-positive diplo- or streptococcus the colonies of which in blood agar show the characteristic green color and are surrounded by a narrow pale green halo. This coccus is not dissolved by bile; in all, 26 cases.

II. Cases 68, 71, 74, 105, 106, as Group I, except that the colonies in blood agar were of a brownish or indifferent color. Case 121 where no growth in blood agar was obtained (here absence of laking

of the red blood cells and diffusion of hemoglobin through the blood-broth mixture would distinguish this growth from that of a typical hemolytic streptococcus).

III. In cases 2, 17, 18, 29, 43, 69 the records are insufficient and the bacteriological diagnosis is made from the clinical type of case.

IV. In cases 26, 39, 129 no bile test was made, otherwise as Group I.

V. Case 73, as Group I, but there is a note of the coccus having been dissolved by bile.

Among these cocci there are only two instances of confusion by means of the bile test. This test as usually performed consisted in the addition of an equal quantity of ox-bile to a convenient amount of a fine suspension of the organism in normal saline. The reaction is quantitative rather than qualitative as a method of differentiating the pneumococcus from the viridans; there are strains of the latter which are dissolved by bile if a relatively large amount of bile is used. The test should be confirmed on several secondary blood agar plants if inconclusive.

Other morphological and cultural characteristics of Gram-positive diplococci are of value in differentiation, especially the presence of capsules; the precipitation of glucose ascitic agar; and the fermentation of inulin, but a classification of the diplococci analyzed in this report by means of any one of these attributes would make a very different list.

So far as animal inoculation is concerned 7 per cent. (cases Nos. 15, 21, 25, 105, 127, 129, 130) of the cocci grouped under viridans were tried on the smaller laboratory animals without producing demonstrated lesions. The relative absence of pathogenicity of the viridans is of great aid in grouping certain doubtful strains of diplococci.

In the accompanying table the more common clinical conditions are summarized. It will be noted that no case with endocarditis recovered, that the prognosis of bacteriemia with otitis or with osteomyelitis is relatively good, while 20.4 per cent. of cases of sepsis recovered. Following Jochmann's[7] definition we have included under sepsis those examples of systemic response to the invasion of the blood-stream by pyogenic organisms or their toxins in which the symptoms arising from the blood infection or intoxication stand in the foreground of the clinical picture as distinguished from bacteriemia, which signifies the mere presence of microorganisms in the blood-stream without reference to symptoms or other conditions.

Summary of Blood Counts. In the following are tabulated the average number of leukocytes and the polynuclear percentage

[7] Loc cit.

of the fatal and of the recovered cases of the commoner varieties of bacteriemia, with a general summary showing the averages of all cases.

GENERAL SUMMARY.

Organism.	Number of cases.	Died.	Recovered.	Improved.	Unimproved.	Died per cent.
Streptococcus hemolyticus	31	21	7	1	2	67.0
Streptococcus viridans	40	25	10	2	3	64.0
Streptococcus mucosus	1	1	..	..	..	100.0
Staphylococcus aureus	39	22	14	3	..	56.0
Staphylococcus albus	3	2	1	..	..	66.0
Pneumococcus	10	6	2	..	2	60.0
Bacillus coli	6	4	2	..	..	66.0
Bacillus influenzæ	2	2	..	..	..	100.0
Anaërobic streptococci	3	2	1	..	..	66.0
Bacillus mucosus capsulatus	1	0	1	..	..	0
Bacillus mallei	2	2	..	..	..	100.0
Bacillus fecalis alkaligenes	1	..	1	..	..	0
Mixed infection	7	6	1	..	..	86.0
Streptococcus hemolyticus and staphylococcus aureus	1	1	..	..	..	100.0
Streptococcus hemolyticus and bacillus typhosus	1	1	..	..	..	100.0
Streptococcus viridans and staphylococcus aureus	1	..	1	..	..	0
Streptococcus viridans and bacillus coli	1	1	..	..	..	100.0
Staphylococcus aureus and bacillus coli	1	1	..	..	..	100.0
Bacillus proteus and bacillus coli	1	1	..	..	..	100.0
Bacillus influenzæ and staphylococcus albus	1	1	..	..	..	100.0
All cases	134	83	..	..	..	61.1

TABLE OF CLINICAL CONDITIONS.

	Died.	Recovered.	Improved.	Unimproved.	Recovered, per cent.
Postpartum infection	10	1	..	1	9.09
Arthritis	1	4	1	..	80.0
Endocarditis	22	..	..	3	0
Otitis media with or without mastoiditis	2	4	..	..	66.6
Pelvic cellulitis	2	..	..	..	0
Osteomyelitis	5	4	1	..	44.4
Sepsis	39	10	3	3	20.4

In general the difference in the blood picture in the fatal and non-fatal cases is not striking. In the average of all cases the former show a slightly higher polynuclear percentage and lower total leukocyte count, but there are exceptions in individual groups. In the streptococcus group, for example, the leukocyte counts in fatal and non-fatal cases are approximately the same, while the polynuclear percentage is the reverse of that anticipated. One may conclude that the ordinary leukocyte and differential blood count is of slight value in prognosis of bacteriemias.

Organism.	Fatal cases. Leukocytes.	Fatal cases. Polynuclears, per cent.	Recovered cases. Leukocytes.	Recovered cases. Polynuclears, per cent.
Streptococcus hemolyticus	18,347	81.4	18,742	85.2
Streptococcus viridans	15,976	78.1	17,222	75.1
Streptococcus mucosus				
Staphylococcus aureus	22,822	85.1	15,380	79.5
Staphylococcus albus	22,800	78.0		
Pneumococcus	26,350	86.5	35,900	82.0
Bacillus coli	18,233	79.0	11,400	70.1
Bacillus influenzæ				
Anaërobic streptococci	18,000	78.0		
Bacillus mucosus capsulatus	..	..	13,800	90.0
Bacillus mallei				
Bacillus fecalis alkaligenes				
Average of all cases	19,877	82.8	17,651	78.3

While it is appreciated that statistics have little value in a series of varied cases such as this, it is of some interest to note the relatively low mortality in the group treated surgically and in the group treated by palliative measures only, and to compare this result with the high mortality in the groups treated by vaccines and sera. Such statistical revelation confirms the impression gained from bedside experience that our present methods of specific therapy of generalized pyogenic infections have not yet established their standing in court.

SUMMARY AND CONCLUSIONS. One hundred and thirty-four examples of bacteriemia from a general hospital service are summarized; 61.1 per cent. of these cases recovered. Noteworthy also is the recovery of 20.4 per cent. of the cases of sepsis. The slight value of the ordinary leukocyte and differential count in prognosis of bacteriemias is shown, as is also the futility of present measures of specific therapy of generalized infections.

RESULTS OF TREATMENT.

Treatment.	Number of cases.	Died.	Recovered	Improved.	Unimproved.	Died per cent.
Vaccines	25	17	4	2	2	81.0
Transfusion	2	2	..	..	..	100.0
Serum	4	3	1	..	..	75.0
Under surgical treatment	47	22	22	3	..	50.0
Palliative	55	39	11	1	4	65.0

STREPTOCOCCUS VIRIDANS—CURED.

CASE No. 88.—*Age*—Thirty-five years. *Diagnosis*—Pulmonary abscess; bacteriemia. *Leukocytes*—21,000; polynuclears, 79 per cent. *Blood culture*—Streptococcus viridans, number not noted (abundant); streptococcus viridans, few; no growth in solid media. *Treatment*—Palliative. *Result*—Cured.

CASE No. 102, D. 5411.—*Age*—Sixteen years. *Diagnosis*—Typhoid fever; phlebitis. *Maximum temperature*—106.4°. *Leuko-*

cytes—10,700; polunuclears, 87 per cent. *Blood culture*—Streptococcus viridans; no growth in solid media. *Treatment*—Palliative. *Result*—Cured.

Case No. 106, D. 4908.—*Age*—Thirty-four years. *Diagnosis*—Acute articular rheumatism; bacteriemia. *Maximum temperature*—103.2°. Leukocytes—19,900; polynuclears, 79 per cent. *Blood culture*—Streptococcus viridans; two positive cultures within a period of several days; no growth in solid media. *Treatment*—Salicylates. *Result*—Cured.

Case No. 130, A. 1719.—*Age*—Twenty-one years. *Diagnosis*—Chronic arthritis of knee; bacteriemia. *Maximum temperature*—99.2°. *Leukocytes*—13,400; polynuclears, 66 per cent. *Blood culture*—Streptococcus viridans; no growth in solid media. *Treatment*—Salicylates. *Result*—Cured.

Case No. 127, A. 2253, A. 2623.—*Age*—Fifty years. *Diagnosis*—Adenocarcinoma of breast; metastasis in axillary fat; sepsis. *Maximum temperature*—103°. *Leukocytes*—9400; polynuclears, 56 per cent. *Blood culture*—Streptococcus viridans; two positive cultures within a period of one week; no growth in solid media; subsequent culture negative. *Treatment*—Vaccines. *Result*—Cured.

Case No. 128, D. 4654.—*Age*—Forty-five years. *Diagnosis*—Sepsis. *Maximum temperature*—105°. *Leukocytes*—24,000; polynuclears, 78 per cent. *Blood culture*—Streptococcus viridans; four positive cultures within a period of ten days, from 2 to 9 per c.c. *Treatment*—Palliative. *Result*—Cured.

Case No. 129, D. 1538.—*Age*—Eighteen years. *Diagnosis*—Subacute arthritis; chronic endocarditis; bacteriemia. *Maximum temperature*—102.6°. *Leukocytes*—20,000; polynuclears, 64 per cent. *Blood culture*—Streptococcus viridans; no growth in solid media. *Treatment*—Salicylates. *Result*—Cured.

Case No. 74.—*Age*—Sixty years. *Diagnosis*—Bronchitis. *Blood culture*—Streptococcus viridans; no growth in solid media. *Treatment*—Palliative. *Result*—Cured.

Case No. 121, A. 1812.—*Age*—Thirty-one years. *Diagnosis*—Chronic otitis; chronic mastoiditis; chronic appendicitis; bacteriemia. *Maximum temperature*—101°. *Leukocytes*—14,200; polynuclears, 81.5 per cent. *Blood culture*—Staphylococcus aureus accompanied by streptococcus viridans; no growth in solid media. *Treatment*—Appendectomy. *Result*—Cured.

Case No. 105, B. 2087.—*Age*—Fourteen years. *Diagnosis*—Acute appendicitis; retrocecal abscess; bacteriemia. *Maximum temperature*—104.6°. *Leukocytes*—22,400; polynuclears, 86 per cent. *Blood culture*—Streptococcus viridans; no growth in solid media. *Treatment*—Incision and drainage of appendicular and subsequent abscess. *Result*—Cured.

STREPTOCOCCUS VIRIDANS—IMPROVED.

CASE No. 21, 1909, VIII, 1.—*Age*—Twelve years. *Diagnosis*—Sepsis. *Maximum temperature*—104°. *Leukocytes*—20,000; polynuclears, 69.5 per cent. *Blood culture*—Streptococcus viridans; no growth in solid media; subsequent cultures negative. *Treatment*—Palliative. *Result*—Improved.

CASE No. 110, E. 360.—*Age*—Thirteen years. *Diagnosis*—Acute articular rheumatism; chronic endocarditis; bacteremia. *Maximum temperature*—104.4°. *Leukocytes*—24,300; polynuclears, 74 per cent. *Blood culture*—Streptococcus viridans: 1 per c.c. *Treatment*—Thoracentesis; salicylates. *Result*—Improved.

STREPTOCOCCUS VIRIDANS—UNIMPROVED.

CASE No. 6, D. 4512.—*Diagnosis*—Acute septic endocarditis. *Maximum temperature*—104°. *Leukocytes*—11,200; polynuclears 71 per cent. *Blood culture*—Streptococcus viridans: 40 per c.c. *Treatment*—Vaccines. *Result*—Unimproved.

CASE 15, D. 1923.—*Age*—Sixteen years. *Diagnosis*—Septic endocarditis. *Maximum temperature*—101.8°. *Leukocytes*—20,800; polynuclears, 43.5 per cent. *Blood culture*—Streptococcus viridans; no growth in solid media. *Treatment*—Salicylates. *Result*—Unimproved.

CASE No. 17, D. 446.—*Age*—Twenty-six years. *Diagnosis*—Chronic + acute endocarditis. *Maximum temperature*—103.8°. *Leukocytes*—32,000; polynuclears, 91 per cent. *Blood culture*—Streptococcus viridans; number of colonies not noted. *Treatment*—Salicylates; vaccines. *Result*—Unimproved.

STREPTOCOCCUS VIRIDANS—DIED.

CASE No. 1, D. 6482.—*Age*—Twenty-eight years. *Diagnosis*—Sepsis; endocarditis. *Maximum temperature*—104°. *Leukocytes*—13,400; polynuclears, 85 per cent. *Blood culture*—Streptococcus viridans; two positive cultures within an interval of a few days; numerous in both colonies. *Treatment*—Palliative. *Result*—Died.

CASE No. 2, D. 6014.—*Age*—Forty years. *Diagnosis*—Acute septic endocarditis. *Maximum temperature*—106.2°. *Leukocytes*—19,200; polynuclears, 73 per cent. *Blood culture*—Streptococcus viridans; two positive cultures within an interval of a few days; numerous colonies in second. *Treatment*—Vaccines. *Result*—Died.

CASE No. 4, D. 4698.—*Age*—Thirty-five years. *Diagnosis*—Sepsis; pericarditis. *Maximum temperature*—100°. *Leukocytes*—7800; polynuclears, 79 per cent. *Blood culture*—Streptococcus viridans; innumerable colonies. *Treatment*—Palliative. *Result*—Died.

CASE No. 7, D. 4359.—*Age*—Fifty-two years. *Diagnosis*—Septic endocarditis. *Maximum temperature*—103°. *Leukocytes*—19,100; polynuclears, 88 per cent. *Blood culture*—Streptococcus viridans; no growth in solid media. *Treatment*—Vaccines. *Result*—Died.

CASE No. 18, D. 243.—*Age*—Twenty years. *Diagnosis*—Sepsis. *Maximum temperature*—105°. *Leukocytes*—33,000; polynuclears, 88.5 per cent. *Blood culture*—Streptococcus viridans, number of colonies not noted. *Treatment*—Salicylates; vaccines. *Result*—Died.

CASE No. 20, D. 5054.—*Age*—Sixty-one years. *Diagnosis*—Sepsis. *Maximum temperature*—104.4°. *Leukocytes*—8000; polynuclears, 54 per cent. *Blood culture*—Streptococcus viridans; two positive cultures within an interval of a few days: 1 per c.c. *Treatment*—Salicylates. *Result*—Died.

CASE No. 25, D. 365.—*Diagnosis*—Acute septic endocarditis. *Maximum temperature*—104.6°. *Leukocytes*—11,600; polynuclears, 72 per cent. *Blood culture*—Streptococcus viridans; three positive cultures within a period of several days, from 80 to 140 per c.c. *Treatment*—Palliative. *Result*—Died.

CASE No. 26, C. 255, D. 605.—*Age*—Forty-two years. *Diagnosis*—Acute septic endocarditis, hydrosalpinx. *Maximum temperature*—104.2°. *Leukocytes*—18,000; polynuclears, 79 per cent. *Blood culture*—Streptococcus viridans; three positive cultures within a period of several days, from 25 to 180 c.c. *Treatment*—Right salpingo-oörphorectomy. *Result*—Died.

CASE No. 30, D. 3695.—*Age*—Twenty-six years. *Diagnosis*—Chronic + acute septic endocarditis. *Maximum temperature*—102.8°. *Leukocytes*—9600; polynuclears, 69 per cent. *Blood culture*—Streptococcus viridans; two positive cultures within a period of a few days; 100 per c.c. in second. *Treatment*—Salicylates. *Result*—Died.

CASE No. 37, C. 2292.—*Age*—Twenty-nine years. *Diagnosis*—Acute diffuse peritonitis; pelvic cellulitis; meningitis; sepsis. *Maximum temperature*—106.6°. *Leukocytes*—19,500; polynuclears, 89 per cent. *Blood culture*—Streptococcus viridans: 2 per c.c., and Bacillus coli (few). *Treatment*—Palliative. *Result*—Died.

CASE No. 39, D. 5355.—*Diagnosis*—Subacute endocarditis. *Maximum temperature*—102°. *Leukocytes*—13,600; polynuclears, 77 per cent. *Blood culture*—Streptococcus viridans; no growth in solid media. *Treatment*—Palliative. *Result*—Died.

CASE No. 40, D. 5726.—*Age*—Twenty years. *Diagnosis*—Sepsis. *Maximum temperature*—104.2°. *Leukocytes*—10,200; polynuclears, 84 per cent. *Blood culture*—Streptococcus viridans; no growth in solid media. *Treatment*—Vaccines. *Result*—Died.

CASE No. 42, D. 6282.—*Diagnosis*—Osteomyelitis of vertebræ; bacteriemia. *Maximum temperature*—103.8°. *Leukocytes*—5600; polynuclears, 72 per cent. *Blood culture*—Streptococcus viridans; no growth in solid media. *Treatment*—Palliative. *Result*—Died.

CASE No. 43, D. 1087.—*Age*—Thirty-one years. *Diagnosis*—Acute septic endocarditis. *Maximum temperature*—104°. *Leukocytes*—16,000; polynuclears, 78 per cent. *Blood culture*—Streptococcus viridans; no growth in solid media. *Treatment*—Palliative. *Result*—Died.

CASE No. 44, D. 1919.—*Age*—Twenty-three years. *Diagnosis*—Chronic + acute endocarditis. *Maximum temperature*—102.6°. *Leukocytes*—12,000. *Blood cultures*—Streptococcus viridans; eight positive cultures within a period of several weeks, from 2 to 70 per c.c. *Treatment*—Palliative. *Result*—Died.

CASE No. 45, D. 3783.—*Age*—Forty-five years. *Diagnosis*—Chronic + acute endocarditis. *Maximum temperature*—104°. *Leukocytes*—13,000; polynuclears, 78 per cent. *Blood cultures*—Streptococcus viridans: 4 per c.c. *Treatment*—Vaccine. *Result*—Died.

CASE No. 49, D. 4936.—*Age*—Thirty-one years. *Diagnosis*—Sepsis. *Maximum temperature*—104.6°. *Leukocytes*—10,500; polynuclears, 49 per cent. *Blood culture*—Streptococcus viridans; two positive cultures within a period of a few days: 15 per c.c. in first, 300 per c.c. in second. *Treatment*—Transfusion. *Result*—Died.

CASE No. 61.—*Age*—Twenty years. *Diagnosis*—Subacute endocarditis; pyelitis. *Blood culture*—Streptococcus viridans, 2 per c.c. *Treatment*—Palliative. *Result*—Died.

CASE No. 68, E. 109, E. 313, E. 420, D. 5084.—*Age*—Ten years. *Diagnosis*—Acute arthritis; sepsis. *Maximum temperature*—104.4°. *Leukocytes*—33,400; polynuclears, 85 per cent. *Blood culture*—Streptococcus viridans; no growth in solid media; culture one month later negative. *Treatment*—Palliative. *Result*—Died.

CASE No. 69.—*Age*—Thirty-five years. *Diagnosis*—Septic endocarditis. *Leukocytes*—24,000; polynuclears, 92 per cent. *Blood culture*—Streptococcus viridans; no growth in solid media. *Treatment*—Vaccines. *Result*—Died.

CASE No. 29, 1909, X, 12.—*Age*—Thirty years. *Diagnosis*—Septic endocarditis. *Maximum temperature*—106.2°. *Leukocytes*—15,000; polynuclears, 88 per cent. *Blood culture*—Streptococcus viridans; three positive cultures within a period of one week; six to twelve colonies per c.c. *Treatment*—Vaccines. *Result*—Died.

CASE No. 70.—*Age*—Sixty years. *Diagnosis*—Chronic + acute endocarditis. *Blood culture*—Streptococcus viridans; no growth in solid media. *Result*—Died.

CASE No. 71.—*Age*—Twenty years. *Diagnosis*—Sepsis. *Blood culture*—Streptococcus viridans: 3 per c.c. *Result*—Died.

CASE No. 73.—*Age*—Thirty years. *Diagnosis*—Subacute endocarditis. *Blood culture*—Streptococcus viridans; no growth in solid media. *Treatment*—Vaccines. *Result*—Died.

CASE No. 91, D. 1671.—*Age*—Thirty-six years. *Diagnosis*—Septic endocarditis. *Maximum temperature*—106°. *Leukocytes*—

23,000; polynuclears, 95 per cent. *Blood culture*—Streptococcus viridans; five positive cultures within a period of a week; 3 or 4 per c.c. *Treatment*—Celiotomy; palliative. *Result*—Died.

STREPTOCOCCUS HEMOLYTICUS—CURED.

Case No. 9, B. 3339.—*Age*—Twenty-eight years. *Diagnosis*—Acute mastoiditis; erysipelas; bacteriemia. *Maximum temperature*—104.8°; *Leukocytes*—22,600; polynuclears, 92 per cent. *Blood culture*—Streptococcus hemolyticus; two positive cultures within a period of a few days, from 8 to 16 per c.c. *Treatment*—Mastoidectomy. *Result*—Cured.

Case No. 19, B. 82.—*Age*—Forty years. *Diagnosis*—Cellulitis of hand and forearm; bacteriemia. *Maximum temperature*—105°. *Leukocytes*—21,000; polynuclears, 81 per cent. *Blood culture*—Streptococcus hemolyticus: 4 per c.c. *Treatment*—Multiple incisions. *Result*—Cured.

Case No. 109, C. 278.—*Age*—Twenty-six years. *Diagnosis*—Pelvic abscess; sepsis. *Maximum temperature*—103.2°. *Leukocytes*—10,700; polynuclears, 77 per cent. *Blood culture*—Streptococcus hemolyticus; no growth in solid media. *Treatment*—Left salpingo-oöphorectomy; posterior colpotomy; drainage; vaccines. *Result*—Cured.

Case No. 123, A. 1495.—*Age*—Twenty-seven years. *Diagnosis*—Scarlet fever; bacteriemia. *Maximum temperature*—105.3°. *Leukocytes*—15,000; polynuclears, 93 per cent. *Blood culture*—Streptococcus hemolyticus; four positive cultures within a period of four days, from 2 to 100 per c.c. *Treatment*—Partial mastoidectomy; phlebotomy of left lateral sinus. *Result*—Cured.

Case No. 124, A. 198.—*Age*—Twenty-four years. *Diagnosis*—Acute otitis media; bacteriemia. *Maximum temperature*—101°. *Leukocytes*—14,900; polynuclears, 78.5 per cent. *Blood culture*—Streptococcus hemolyticus: 6 per c.c. *Treatment*—Paracentesis. *Result*—Cured.

Case No. 125, A. 370.—*Age*—Thirty-two years. *Diagnosis*—Septic infarcts of kidney, sepsis. *Maximum temperature*—104.6°. *Leukocytes*—15,000; polynuclears, 83 per cent. *Blood culture*—Streptococcus hemolyticus, two positive cultures on following days; 5 per c.c. in second. *Treatment*—Palliative. *Result*—Cured.

Case No. 126, A. 5223.—*Age*—Twenty years. *Diagnosis*—Abscess of breast; bacteriemia. *Maximum temperature*—106.3°. *Leukocytes*—32,000; polynuclears, 93 per cent. *Blood culture*—Streptococcus hemolyticus: 3.5 per c.c.; two negative cultures during the following six days. *Treatment*—Multiple incisions and drainage. *Results*—Cured.

STREPTOCOCCUS HEMOLYTICUS—IMPROVED.

CASE No. 13, B. 2492.—*Age*—Twenty-one years. *Diagnosis*—Gunshot wound of chest; bacteriemia. *Maximum temperature*—105°. *Leukocytes*—26,000; polynuclears, 90 per cent. *Blood culture*—Streptococcus hemolyticus; two positive cultures within a period of a few days; no growth in solid media in either. *Treatment*—Tetanus antitoxin; vaccines. *Result*—Improved.

STREPTOCOCCUS HEMOLYTICUS—UNIMPROVED.

CASE No. 41, D. 6225.—*Age*—Nineteen years. *Diagnosis*—Acute tonsillitis; sepsis. *Maximum temperature*—105.4°. *Leukocytes*—17,800; polynuclears, 97 per cent. *Blood culture*—Streptococcus hemolyticus, number of colonies not noted. *Treatment*—Palliative. *Result*—Unimproved.

CASE No. 54, C. 517.—*Age*—Twenty-two years. *Diagnosis*—Incomplete abortion; sepsis. *Maximum temperature*—103°. *Blood culture*—Streptococcus hemolyticus: 110 per c.c. *Treatment*—Palliative. *Result*—Unimproved.

STREPTOCOCCUS HEMOLYTICUS—DIED.

CASE No. 5, A. 3785.—*Age*—Thirty years. *Diagnosis*—Cellulitis of forearm; bacteriemia. *Maximum temperature*—106.3°. *Blood culture*—Streptococcus hemolyticus: 2 per c.c. *Treatment*—Incision and drainage. *Result*—Died.

CASE No. 10, D. 4165.—*Age*—Twelve years. *Diagnosis*—Acute septic endocarditis. *Maximum temperature*—106.8°. *Leukocytes*—9600; polynuclears, 69 per cent. *Blood culture*—Streptococcus hemolyticus, 110 per c.c. *Treatment*—Palliative. *Result*—Died.

CASE No. 14, D. 1850.—*Age*—Eighteen years. *Diagnosis*—Pseudoleukemia; sepsis. *Maximum temperature*—103.8°. Leukocytes—1400; polynuclears, 71.6 per cent. *Blood culture*—Streptococcus hemolyticus; two positive cultures within an interval of a few days; no growth in solid media. *Treatment*—Salvarsan, intravenously. *Result*—Died.

CASE No. 11, B. 2573.—*Diagnosis*—Cancer of breast; infection of wound; sepsis. *Maximum temperature*—105.2°. *Leukocytes*—16,200; polynuclears, 87 per cent. *Blood culture*—Streptococcus hemolyticus; two positive cultures within a period of a few days: 1 per c.cm in first; few colonies of streptococcus in second, accompanied by Staphylococcus pyogenes aureus. *Treatment*—Complete mastectomy, with removal of pectoral muscles and axillary lymph glands. *Result*—Died.

CASE No. 12, C. 1476.—*Age*—Twenty-two years. *Diagnosis*—Ectopic gestation; rupture; bacteriemia. *Maximum temperature*—

100°. *Leukocytes*—16,400; polynuclears, 82 per cent. *Blood culture*—Streptococcus hemolyticus, innumerable colonies. *Treatment*—Partial hysterectomy. *Result*—Died.

CASE No. 16, D. 1358.—*Age*—Twenty months. *Diagnosis*—Otitis media; sepsis. *Maximum temperature*—107.6°. *Blood culture*—Streptococcus hemolyticus, 120 per c.c. *Treatment*—Palliative. *Result*—Died.

CASE No. 22, A. 335.—*Diagnosis*—Complete abortion; sepsis. *Maximum temperature*—104.6°. *Leukocytes*—27,400; polynuclears, 91 per cent. *Blood culture*—Streptococcus hemolyticus, colonies few, number not noted. *Treatment*—Vaccines. *Results*—Died.

CASE No. 23, B. 11.—*Age*—Nineteen years. *Diagnosis*—Acute salpingitis; peritonitis; pneumonia; sepsis. *Maximum temperature*—105.6°. *Leukocytes*—22,800; polynuclears, 87 per cent. *Blood culture*—Streptococcus hemolyticus, abundant growth; number of colonies not noted. *Treatment*—Palliative. *Result*—Died.

CASE No. 27, 1909, III, 8.—*Age*—Twenty-five years. *Diagnosis*—Puerperal sepsis. *Maximum temperature*—105.8°. *Leukocytes*—11,200; polynuclears, 88 per cent. *Blood culture*—Streptococcus hemolyticus; two positive cultures within a period of a few days: 8 per c.c. in first; number not noted in second. *Treatment*—Vaccines; serum. *Result*—Died.

CASE No. 32, C. 383.—*Age*—Thirty-two years. *Diagnosis*—Pelvic cellulitis; polyarthritis; sepsis. *Maximum temperature*—104.4°. *Leukocytes*—9400; polynuclears, 76 per cent. *Blood culture*—Streptococcus hemolyticus; three positive cultures within a period of three or four days, three to seven colonies per c.c. *Treatment*—Incision and drainage of abscess of knees. *Result*—Died.

CASE No. 33, C. 667.—*Age*—Eighteen years. *Diagnosis*—Postpartum sepsis. *Maximum temperature*—105°. *Leukocytes*—21,000; polynuclears, 86 per cent. *Blood culture*—Streptococcus hemolyticus: 12 per c.c. *Treatment*—Serum. *Result*—Died.

CASE No. 38, D. 4589.—*Age*—Thirty-one years. *Diagnosis*—Chronic + acute endocarditis; chronic nephritis; chronic pulmonary tuberculosis. *Maximum temperature*—106°. *Leukocytes*—35,100; polynuclears, 92 per cent. *Blood cultures*—Streptococcus hemolyticus: 110 per c.c. *Treatment*—Palliative. *Result*—Died.

CASE No. 46, D. 3825.—*Age*—Fifty-seven years. *Diagnosis*—Sepsis. *Maximum temperature*—106.6°. *Leukocytes*—4400; polynuclears, 85 per cent. *Blood culture*—Streptococcus hemolyticus: 100 per c.c. *Treatment*—Palliative. *Result*—Died.

CASE No. 47, D. 4914.—*Age*—Twenty-one years. *Diagnosis*—Acute lymphatic leukemia; sepsis. *Maximum temperature*—105.6°. *Leukocytes*—28,900; polynuclears, 1 per cent. *Blood culture*—Streptococcus hemolyticus; only fluid media used. *Treatment*—Palliative. *Result*—Died.

CASE No. 48, D. 4946.—*Diagnosis*—Sepsis. *Maximum temperture*—105°. *Leukocytes*—7,200; polynuclears, 92 per cent. *Blood culture*—Streptococcus hemolyticus; two positive cultures on following days, 360 per c.c. in latter. *Treatment*—Palliative. *Result*—Died.

CASE No. 50, C. 2918.—*Age*—Thirty-six years. *Diagnosis*—Complete abortion; sepsis. *Maximum temperature*—108°. *Leukocytes*—17,200; polynuclears, 93 per cent. *Blood culture*—Streptococcus hemolyticus, number of colonies not noted. *Treatment*—Palliative. *Result*—Died.

CASE No. 51, B. 5597, C. 4816.—*Age*—Thirty-four years. *Diagnosis*—Acute endometritis; thrombophlebitis of pelvic veins; sepsis; *Maximum temperature*—105.6°. *Leukocytes*—4400; polynuclears, 80 per cent. *Blood culture*—Streptococcus hemolyticus; four positive cultures within a period of two weeks, from 1 to 100 per c.c. *Treatment*—Serum; transfusion; pelvic drainage. *Result*—Died.

CASE No. 55, C. 2044.—*Age*—Twenty-two years. *Diagnosis*—Incomplete abortion; sepsis. *Maximum temperature*—106.2°. *Leukocytes*—12,000; polynuclears, 96 per cent. *Blood culture*—Streptococcus hemolyticus: 250 per c.c. *Treatment*—Vaccines. *Result*—Died.

CASE No. 89, D. 1116.—*Age*—Ten years. *Diagnosis*—Typhoid fever; streptococcus; bacteriemia. *Maximum temperature*—106.2°. *Leukocytes*—15,000; polynuclears, 88 per cent. *Blood culture*—Bacillus typhosus and Streptococcus hemolyticus; only fluid media used. *Treatment*—Typhoid; salicylates. *Result*—Died.

CASE No. 24, C. 114.—*Age*—Twenty-nine years. *Diagnosis*—Abscess of uterus; sepsis. *Maximum temperature*—101.6°. *Leukocytes*—67,000; polynuclears, 95 per cent. *Blood culture*—Streptococcus hemolyticus, number of colonies not noted. *Treatment*—Complete hysterectomy. *Result*—Died.

CASE No. 28, 1909, IX, 11.—*Age*—Thirty-eight years. *Diagnosis*—Sepsis. *Maximum temperature*—106.4°. *Leukocytes*—22,000; polynuclears, 84 per cent. *Blood culture*—Streptococcus hemolyticus; number of colonies not noted. *Treatment*—Curettage; bilateral trachelorrhaphy. *Result*—Died.

STREPTOCOCCUS MUCOSUS—DIED.

CASE No. 36, A. 2498.—*Age*—Fourteen years. *Diagnosis*—Fracture of base; suppurative meningitis; sepsis. *Maximum temperature*—106°. *Leukocytes*—19,000; polynuclears, 81 per cent. *Blood culture*—Streptococcus mucosus, 1 per c.c. *Treatment*—Vaccines. *Result*—Died.

STAPHYLOCOCCUS AUREUS—CURED.

CASE No. 59.—*Diagnosis*—Osteomyelitis of tibia; bacteriemia. *Blood culture*—Staphylococcus aureus: 1 per c.c. *Treatment*—Vaccines, amputation. *Result*—Cured.

CASE No. 60.—*Age*—Fifty years. *Diagnosis*—Panophthalmitis; frontal sinusitis; bacteriemia. *Blood culture*—Staphylococcus aureus: 1 per c.c. two days after operation. *Treatment*—Operation. *Result*—Cured.

CASE No. 97, A. 1670.—*Age*—Twenty-five years. *Diagnosis*—Infected wound of nose; bacteriemia. *Maximum temperature*—102.8°. *Leukocytes*—16,600; polynuclears, 70 per cent. *Blood culture*—Staphylococcus aureus, no growth in solid media. *Treatment*—Palliative. *Result*—Cured.

CASE No. 111, A. 1695.—*Age*—Sixteen years. *Diagnosis*—Streptococcus; abscess of deep cervical glands; staphylococcus; bacteriemia. *Maximum temperature*—103.4°. *Leukocytes*—9000; polynuclears, 72 per cent. *Blood culture*—Staphylococcus aureus; no growth in solid media. *Treatment*—Excision of lymph nodes on left side of neck. *Result*—Cured.

CASE No. 112, B. 3967.—*Age*—Forty-two years. *Diagnosis*—Cellulitis of leg; bacteriemia. *Maximum temperature*—106°. *Blood culture*—Staphylococcus aureus; no growth in solid media; culture negative the following day and again three weeks later. *Treatment*—Multiple incisions and drainage. *Result*—Cured.

CASE No. 113, B. 4517.—*Diagnosis*—Sepsis. *Maximum temperature*—102.3°. *Leukocytes*—11,000; polynuclears, 72 per cent. *Blood culture*—Staphylococcus aureus; two positive cultures within a period of two days; no growth in solid media. *Treatment*—Palliative. *Result*—Cured.

CASE No. 114, A. 5096.—*Age*—Nine years. *Diagnosis*—Osteomyelitis of left tibia and of right radius; suppurative arthritis of right knee; sepsis. *Maximum temperature*—103.8°. *Leukocytes*—22,600; polynuclears, 84 per cent. *Blood culture*—Staphylococcus aureus; no growth in solid media. *Treatment*—Amputation of leg, partial ostectomy of right radius, incision and drainage of right patella; vaccines. *Result*—Cured.

CASE No. 115, A. 4815.—*Age*—Eighteen years. *Diagnosis*—Chronic suppurative osteomyelitis of humerus; bacteriemia. *Maximum temperature*—104°. *Leukocytes*—25,000; polynuclears, 88 per cent. *Blood culture*—Staphylococcus aureus; no growth in solid media. *Treatment*—Incision of outer aspect of lower third of arm; sequestrum removed. *Result*—Cured.

CASE No. 116, A. 4763.—*Age*—Thirty-two years. *Diagnosis*—Chronic suppurative osteomyelitis of tibia; bacteriemia. *Maximum temperature*—102.4°. *Blood culture*—Staphylococcus aureus;

no growth in solid media. *Treatment*—Sequestrotomy; partial osteotomy of tibia. *Result*—Cured.

Case No. 117, D. 2058, B. 1954.—*Age*—Thirty-three years. *Diagnosis*—Subphrenic abscess; multiple skin abscesses; sepsis. *Maximum temperature*—102.8°. *Leukocytes*—11,000; polynuclears, 72 per cent. *Blood culture*—Staphylococcus aureus: 1 per c.c., culture one week later negative. *Treatment*—Appendix removed; abscess drained. *Result*—Cured.

Case No. 118, C. 2457.—*Age*—Fifty-five years. *Diagnosis*—Left hydrosalpinx; bacteriemia. *Maximum temperature*—101°. *Leukocytes*—13,000; polynuclears, 88 per cent. *Blood culture*—Staphylococcus aureus; no growth in solid media. *Treatment*—Left salpingo-oöphorectomy. *Result*—Cured.

Case No. 119, C. 4413.—*Age*—Twenty-six years. *Diagnosis*—Peritoneal abscess (pelvic) bacteriemia. *Maximum temperature*—104°. *Leukocytes*—16,000; polynuclears, 90 per cent. *Blood cultures*—Staphylococcus aureus; no growth in solid media; there were two negative cultures, one preceding the other following the positive culture by one month. *Treatment*—Posterior colpotomy. *Result*—Cured.

Case 121, A. 1812.—*Age*—Thirty-one years. *Diagnosis*—Chronic otitis; chronic mastoiditis; chronic appendicitis, bacteriemia. *Maximum temperature*—101°. *Leukocytes*—14,200; polynuclears, 81.5 per cent. *Blood cultures*—Staphylococcus aureus; no growth in solid media accompanied by Streptococcus viridans. *Treatment*—Appendectomy. *Result*—Cured.

Case No. 122, D. 3407.—*Diagnosis*—Sepsis. *Maximum temperature*—105°. *Leukocytes*—15,400; polynuclears, 78 per cent. *Blood culture*—Staphylococcus aureus; three positive cultures within a period of twenty-five days: 1 per c.c. largest number of colonies. *Treatment*—Palliative. *Result*—Cured.

STAPHYLOCOCCUS AUREUS—IMPROVED.

Case No. 77, A. 5775.—*Age*—Twelve years. *Diagnosis*—Osteomyelitis of humerus; bacteriemia. *Maximum temperature*—104°. *Leukocytes*—18,000; polynuclears, 84 per cent. *Blood culture*—Staphylococcus aureus; three positive cultures within a period of a week; no growth in solid media. *Treatment*—Osteotomy, vaccines. *Result*—Improved.

Case No. 80, D. 3975.—*Age*—Fifteen years. *Diagnosis*—Chronic endocarditis; sepsis. *Maximum temperature*—102°. *Leukocytes*—11,400; polynuclears, 79 per cent. *Blood culture*—Staphylococcus aureus; no growth in solid media; culture four days later negative. *Treatment*—Salicylates. *Result*—Improved.

Case No. 133, A. 1941.—*Age*—Fifty years. *Diagnosis*—Prostatic hypertrophy; sepsis. *Maximum temperature*—104°. *Leuko-*

cytes—12,000; polynuclears, 77 per cent. *Blood culture*—Staphylococcus aureus, number of colonies not noted; probably an instance of bacteriemia of urethral chill. *Treatment*—Prostatectomy. *Result*—Improved.

STAPHYLOCOCCUS AUREUS—DIED.

CASE No. 11, B. 2573.—*Diagnosis*—Cancer of breast; infection of wound; sepsis. *Maximum temperature*—105.2°. *Leukocytes*—16,200; polynuclears, 87 per cent. *Blood culture*—Staphylococcus aureus, accompanied by a few colonies of Streptococcus hemolyticus. *Treatment*—Complete mastectomy, with removal of pectoral muscles and axillary lymph glands. *Result*—Died.

CASE No. 31, 1909, XII, 11.—*Age*—Fourteen years. *Diagnosis*—Acute epiphysitis of hip; bacteriemia. *Maximum temperature*—106.2°. *Leukocytes*—14,000; polynuclears, 80.5 per cent. *Blood culture*—Staphylococcus aureus; two positive cultures within a period of several days; number of colonies not noted. *Treatment*—Incision and drainage of abscess about hip; vaccines. *Result*—Died.

CASE No. 56, B. 3814.—*Age*—Sixteen years. *Diagnosis*—Osteomyelitis; suppurative arthritis; sepsis. *Maximum temperature*—104.2°. *Leukocytes*—30,000; polynuclears, 88 per cent. *Blood culture*—Staphylococcus aureus, two positive cultures within a period of several weeks; 3 per c.c. in first, 1 per c.c. in second. *Treatment*—Incision of knee. *Result*—Died.

CASE No. 57, A. 3683.—*Age*—Eleven years. *Diagnosis*—Multiple abscesses; sepsis. *Maximum temperature*—104°. *Leukocytes*—37,200; polynuclears, 87 per cent. *Blood culture*—Staphylococcus aureus; no growth in solid media. *Treatment*—Incision and drainage of abscess of thigh. *Result*—Died.

CASE No. 62, D. 6438, A. 5346.—*Age*—Thirty-nine years. *Diagnosis*—Lumbar abscess; sepsis. *Maximum temperature*—105.6°. *Leukocytes*—16,000; polynuclears, 86 per cent. *Blood culture*—Staphylococcus aureus; two positive cultures within a period of a few days: 9 per c.c. in first, numerous in second. *Treatment*—Exploration of lumbar region. *Result*—Died.

CASE No. 63, E. 317, B. 5317, B. 5336.—*Age*—Two years, eight months. *Diagnosis*—Osteomyelitis of rib; sepsis. *Maximum temperature*—105°. *Leukocytes*—24,000; polynuclears, 91 per cent. *Blood culture*—Staphylococcus aureus: many per c.c. *Treatment*—Palliative. *Result*—Died.

CASE No. 64, D. 4707.—*Age*—Thirty-nine years. *Diagnosis*—Pulmonary tuberculosis; sepsis. *Maximum temperature*—105.8°. *Leukocytes*—19,900; polynuclears, 86 per cent. *Blood culture*—Staphylococcus aureus: 300 per c.c.; culture about six weeks, later negative. *Treatment*—Palliative. *Result*—Died.

CASE No. 65, 1909, VII, 30.—*Age*—Thirty-eight years. *Diagnosis*—Chronic endocarditis. *Maximum temperature*—104.6°. *Leukocytes*—17,500; polynuclears, 86 per cent. *Blood culture*—Staphylococcus aureus: 1 per c.c.; culture few days earlier negative. *Treatment*—Palliative. *Result*—Died.

CASE No. 66, D. 6258.—*Age*—Thirty-eight years. *Diagnosis*—Phlebitis of portal vein; pericarditis; chronic endocarditis; sepsis. *Maximum temperature*—107°. *Leukocytes*—10,000; polynuclears, 91 per cent. *Blood culture*—Staphylococcus aureus: several colonies per c.c. *Treatment*—Palliative. *Result*—Died.

CASE No. 76, B. 4336.—*Age*—Twenty-nine years. *Diagnosis*—Cholelithiasis; chronic appendicitis; sepsis. *Maximum temperature*—107°. *Blood culture*—Staphylococcus aureus + Bacillus coli; no growth in solid media. *Treatment*—Cholecystectomy; appendectomy. *Result*—Died.

CASE No. 78.—*Age*—Thirty years. *Diagnosis*—Typhoid fever; sepsis. *Blood culture*—Staphylococcus aureus; no growth in solid media. *Treatment*—Typhoid. *Result*—Died.

CASE No. 79, D. 3214.—*Age*—Forty-seven years. *Diagnosis*—Chronic endocarditis; sepsis. *Maximum temperature*—105°. *Leukocytes*—15,000; polynuclears, 89 per cent. *Blood culture*—Staphylococcus aureus; two positive cultures on following days: in each innumerable colonies. *Treatment*—Palliative. *Report*—Died.

CASE No. 81, B. 3479.—*Age*—Fifteen years. *Diagnosis*—Osteomyelitis; sepsis. *Maximum temperature*—105.6°. *Leukocytes*—13,600; polynuclears, 87 per cent. *Blood culture*—Staphylococcus aureus, two positive cultures on following days: 100 per c.c. in first, 15 per c.c. in second. *Treatment*—Incision and drainage of cellulitis of thigh; vaccines. *Result*—Died.

CASE No. 82, D. 4356.—*Age*—Thirty-nine years. *Diagnosis*—Meningitis; sepsis. *Maximum temperature*—106.6°. *Leukocytes*—50,000; polynuclears, 65.8 per cent. *Blood culture*—Staphylococcus aureus: 60 per c.c. *Treatment*—Palliative. *Result*—Died.

CASE No. 83, A. 4039.—*Age*—Seventeen years. *Diagnosis*—Osteomyelitis; arthritis; sepsis. *Maximum temperature*—106.4°. *Leukocytes*—18,000; polynuclears, 89 per cent. *Blood culture*—Staphylococcus aureus; three positive cultures within a period of a few days: 10 per c.c. in last one. *Treatment*—Partial ostectomy of tibia with drainage; arthrotomy with drainage. *Result*—Died.

CASE No. 84.—*Age*—Fifty years. *Diagnosis*—Femoral phlebitis: colitis: sepsis. *Maximum temperature*—101°. *Blood culture*—Staphylococcus aureus: no growth in solid media. *Treatment*—Palliative. *Result*—Died.

CASE No. 85, B. 4564.—*Diagnosis*—Septic infarct of kidney; acute nephritis; sepsis. *Maximum temperature*—102.4°. *Leukocytes*—18,000; polynuclears, 89 per cent. *Blood culture*—Staphylococcus aureus; abundant growth; number not noted. *Treatment*—Palliative. *Result*—Died.

CASE No. 93, 1909, VI, 7.—*Age*—Forty-eight years. *Diagnosis*—Sepsis. *Maximum temperature*—107.8°. *Leukocytes*—33,000; polynuclears, 85.5 per cent. *Blood culture*—Staphylococcus aureus; abundant growth; number not noted; Staphylococcus aureus from abscess of back. *Treatment*—Palliative. *Result*—Died.

CASE No. 94, 1909, XII, 11.—*Age*—Fourteen years. *Diagnosis*—Acute epiphysitis of hip; sepsis. *Maximum temperature*—106.2°. *Leukocytes*—14,000; polynuclears, 80.5 per cent. *Blood culture*—Staphylococcus aureus: 12 per c.c. *Treatment*—Incision and drainage of abscess about hip; vaccines. *Result*—Died.

CASE No. 95, D. 163.—*Age*—Twenty years. *Diagnosis*—Sepsis. *Maximum temperature*—107.8°. *Leukocytes*—42,000; polynuclears, 85 per cent. *Blood culture*—Staphylococcus aureus; abundant growth; number not noted. *Treatment*—Palliative. *Result*—Died.

CASE No. 96, B. 763.—*Age*—Twenty-six years. *Diagnosis*—Cellulitis of foot; pleurisy; sepsis. *Maximum temperature*—107.4°. *Leukocytes*—21,600; polynuclears, 89 per cent. *Blood culture*—Staphylococcus aureus; abundant growth; number not noted. *Treatment*—Incision; palliative. *Result*—Died.

CASE No. 86.—*Diagnosis*—Chronic endocarditis; acute prostatitis; infarct of kidney; sepsis. *Maximum temperature*—103°. *Blood culture*—Staphylococcus aureus: 30 per c.c. *Result*—Died.

STAPHYLOCOCCUS ALBUS—CURED.

CASE No. 120, D. 1030.—*Age*—Forty-eight years. *Diagnosis*—Typhoid fever; acute orchitis and epididymitis; bacteriemia. *Maximum temperature*—105.6°. *Leukocytes*—5,700; polynuclears, 51 per cent. *Blood culture*—Staphylococcus albus; abundant growth; number not noted. *Treatment*—Palliative. *Result*—Cured.

STAPHYLOCOCCUS ALBUS—DIED.

CASE No. 58, D. 1812.—*Age*—Thirty-six years. *Diagnosis*—Chronic and acute endocarditis. *Maximum temperature*—103.6°. *Leukocytes*—35,600. *Blood culture*—Staphylococcus albus; four positive cultures within a period of a few days; no growth in solid media; Bacillus influenzæ accompanied Staphylococcus albus in each instance. *Treatment*—Palliative. *Result*—Died.

CASE No. 92, 1909, V, 7.—*Age*—Fifty. *Diagnosis*—Septic endocarditis. *Maximum temperature*—104.8°. *Leukocytes*—10,000; polynuclears, 78 per cent. *Blood culture*—Staphylococcus albus; three positive cultures within a period of two or three weeks: 5 per c.c. largest number of colonies. *Treatment*—Vaccines. *Result*—Died.

PNEUMOCOCCUS—CURED.

CASE No. 103, D. 5360.—*Age*—Thirty-eight years. *Diagnosis*—Typhoid fever; lobar pneumonia. *Maximum temperature*—105.2°. *Leukocytes*—8600; polynuclears, 66 per cent. *Blood culture*—Pneumococcus; no growth in solid media. *Treatment*—Palliative. *Result*—Cured.

CASE No. 107, D. 6041.—*Age*—Forty-seven years. *Diagnosis*—Suppurative pericarditis; lobar pneumonia; suppurative pleurisy; arthritis of right wrist; sepsis. *Maximum temperature*—103.2°. *Leukocytes*—63,200; polynuclears, 98 per cent. *Blood culture*—Pneumococcus; no growth in solid media; culture a few days later negative. *Treatment*—Serum intravenously; incision and drainage of pericardium and pleura. *Result*—Cured.

PNEUMOCOCCUS—UNIMPROVED.

CASE No. 3, D. 4909.—*Age*—Thirty-one years. *Diagnosis*—Sepsis. *Maximum temperature*—105.4°. *Leukocytes*—9,800; polynuclears, 77 per cent. *Blood culture*—Pneumococcus; two positive cultures within a period of a few days. *Treatment*—Palliative. *Result*—Unimproved.

CASE No. 8, D. 4142.—*Age*—Four years. *Diagnosis*—Bronchopneumonia; otitis media. *Maximum temperature*—106°. *Leukocytes*—14,000; polynuclears, 77 per cent. *Blood culture*—Pneumococcus; colonies few; number not noted. *Treatment*—Incision of ear drum. *Result*—Unimproved.

PNEUMOCOCCUS—DIED.

CASE No. 34, D. 1689.—*Age*—Eight years. *Diagnosis*—Chronic and acute endocarditis; meningitis. *Maximum temperature*—108°. *Leukocytes*—21,000; polynuclears, 89 per cent. *Blood culture*—Pneumococcus; colonies abundant; number not noted. *Treatment*—Salicylates. *Result*—Died.

CASE No. 35, D. 1780.—*Age*—Thirty-eight years. *Diagnosis*—Chronic otitis media; sepsis. *Maximum temperature*—106.2°. *Leukocytes*—28,300; polynuclears, 94 per cent. *Blood culture*—Pneumococcus: 2 per c.c. *Treatment*—Palliative. *Result*—Died.

CASE No. 100, B. 2339.—*Age*—Forty-eight years. *Diagnosis*—Acute appendicitis; peritoneal abscess; sepsis. *Maximum temperature*—106°. *Leukocytes*—9800; polynuclears, 79 per cent. *Blood culture*—Pneumococcus: 4 per c.c. *Treatment*—Appendectomy with drainage; transpleural hepatotomy. *Result*—Died.

CASE No. 101, A. 3767.—*Age*—Thirty-two years. *Diagnosis*—Abscess of brain; sepsis. *Maximum temperature*—105.8°. *Leukocytes*—18,000; polynuclears, 90 per cent. *Blood culture*—Pneumo-

coccus; innumerable colonies. *Treatment*—Palliative. *Result*—Died.

Case No. 104, E. 251.—*Age*—Three months. *Diagnosis*—Pneumococcus meningitis. *Maximum temperature*—105.4°. *Leukocytes*—17,000; polynuclears, 77 per cent. *Blood culture*—Pneumococcus; innumerable colonies. *Treatment*—Serum. *Result*—Died.

Case No. 72, B. 1197.—*Age*—Sixty-two years.—*Diagnosis*—Sepsis; empyema. *Maximum temperature*—104.2° *Leukocytes*—64,000; polynuclears, 92 per cent. *Blood culture*—Pneumococcus; no growth in solid media. *Treatment*—Thoracotomy. *Result*—Died.

BACILLUS COLI—CURED.

Case No. 75, A. 3142.—*Age*—Twenty-eight years. *Diagnosis*—Mastoiditis; bacteriemia. *Maximum temperature*—106°. *Leukocytes*—15,000; polynuclears, 81 per cent. *Blood culture*—Bacillus coli; 1 per c.c. *Treatment*—Partial mastoidectomy. *Result*—Cured.

Case No. 87, D. 957.—*Age*—Thirty-four years. *Diagnosis*—Gastro-enteritis; bacteriemia. *Maximum temperature*—102.4°. *Leukocytes*—7800; polynuclears, 60 per cent. *Blood culture*—Bacillus coli; no growth in solid media. *Treatment*—Palliative. *Result*—Cured.

BACILLUS COLI—DIED.

Case No. 37, C. 2292.—*Age*—Twenty-nine years. *Diagnosis*—Acute diffuse peritonitis; pelvic cellulitis; meningitis; sepsis. *Maximum temperature*—106.6°. *Leukocytes*—19,500; polynuclears, 89 per cent. *Blood culture*—Bacillus coli; few with Streptococcus viridans. *Treatment*—Palliative. *Result*—Died.

Case No. 90, D. 3427, D. 2037.—*Age*—Three years. *Diagnosis*—Raynaud's disease; bacteriemia. *Maximum temperature*—105.2°. *Leukocytes*—26,400; polynuclears, 71 per cent. *Blood culture*—Bacillus coli, only fluid media used. *Treatment*—Amputation of right arm, two fingers, and right leg. *Result*—Died.

Case No. 98, C. 2200.—*Age*—Fifty-three years. *Diagnosis*—Carcinoma of cervix; sepsis, postoperative. *Maximum temperature*—103.8°. *Leukocytes*—8800; polynuclears, 77 per cent. *Blood culture*—Bacillus coli; two positive cultures: 15 per c.c.; Bacillus coli in first, Bacillus coli and Bacillus proteus the following day; no growth in solid media. *Treatment*—Complete hysterectomy. *Result*—Died.

Case No. 76, B. 4336.—*Age*—Twenty-nine years. *Diagnosis*—Cholelithiasis; chronic appendicitis; sepsis. *Maximum temperature*—107°. *Blood culture*—Bacillus coli and aureus; no growth in solid media. *Treatment*—Cholecystectomy, appendectomy. *Result*—Died.

BACILLUS INFLUENZÆ—DIED.

CASE NO. 58, D. 1812.—*Age*—Thirty-six years. *Diagnosis*—Chronic + acute endocarditis. *Maximum temperature*—103.6°. *Leukocytes*—35,600 per cent. *Blood culture*—Bacillus influenzæ; four positive cultures within a period of a few days; no growth in solid media; Staphylococcus albus accompanied Bacillus influenzæ in each instance. *Treatment*—Palliative. *Result*—Died.

CASE NO. 67.—*Age*—Thirty years. *Diagnosis*—Subacute endocarditis. *Blood culture*—Bacillus influenzæ; no growth in solid media. *Treatment*—Palliative. *Result*—Died.

ANAËROBIC STREPTOCOCCI—CURED.

CASE NO. 131, C. 2238.—*Age*—Thirty-two years. *Diagnosis*—Pyosalpinx; pelvic abscess; thrombophlebitis of left femoral; bacteriemia. *Maximum temperature*—105.2°. *Leukocytes*—28,400; polynuclears, 89 per cent. *Blood culture*—Anaërobic streptococci; no growth in solid media. *Treatment*—Posterior colpotomy with drainage. *Result*—Cured.

ANAËROBIC STREPTOCOCCI—DIED.

CASE NO. 52, D. 3900.—*Age*—Thirty years. *Diagnosis*—Acute endometritis; sepsis. *Maximum temperature*—104.6°. *Leukocytes*—10,600; polynuclears, 70 per cent. *Blood culture*—Anaërobic streptococci; no growth in solid media. *Treatment*—Palliative. *Result*—Died.

CASE NO. 53, C. 1915.—*Age*—Forty years. *Diagnosis*—Pyosalpinx; sepsis. *Maximum temperature*—102.6°. *Leukocytes*—25,400; polynuclears, 86 per cent. *Blood culture*—Anaërobic streptococci; no growth in solid media; an anaerobic streptococcus and Bacillus coli obtained from culture of pus from left uterine tube. *Treatment*—Complete abdominal hysterectomy; right and left salpingo-oöphorectomy; appendectomy; division of adhesions; enterorrhaphy. *Result*—Died.

BACILLUS MUCOSUS CAPSULATUS—CURED.

CASE NO. 108, D. 6526.—*Age*—Forty-five years. *Diagnosis*—Sepsis. *Maximum temperature*—106.3°. *Leukocytes*—13,800; polynuclears, 90 per cent. *Blood culture*—Bacillus mucosus capsulatus; three positive cultures within a period of four days; growth in fluid media in all; in one only a growth in solid media of 3 or 4 colonies per c.c. Three negative cultures during the following six weeks. *Treatment*—Fixation abscess; incision and drainage of right leg. *Result*—Cured.

BACILLUS MALLEI—DIED.

CASE No. 99, D. 1140.—*Age*—Thirty-eight years. *Diagnosis*—Glanders. *Maximum temperature*—105.6°. *Blood culture*—Bacillus mallei; no growth in solid media. *Treatment*—Palliative. *Result*—Died.

CASE No. 132, D. 1737.—*Age*—Thirty-seven years. *Diagnosis*—Glanders, balanoposthitis. *Maximum temperature*—105.4°. *Leukocytes*—26,000; polynuclears, 87 per cent. *Blood culture*—Bacillus mallei; no growth in solid media. *Treatment*—Palliative. *Result*—Died.

BACILLUS FECALIS ALKALIGENES—CURED.

CASE No. 134.—*Age*—Thirty years. *Diagnosis*—Gastroenteritis. *Blood culture*—Bacillus fecalis alkaligenes; no growth in solid media. *Treatment*—Palliative. *Result*—Cured.

DIABETES IN CHINA.

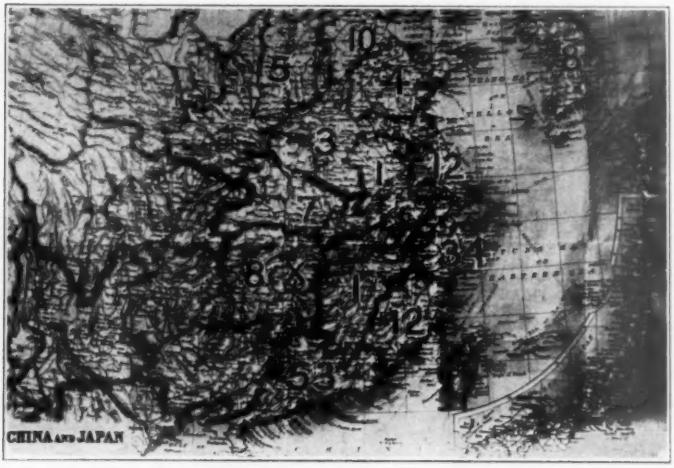
BY ALFRED C. REED, M.D.,
YALE HOSPITAL, CHANGSHA, CHINA.

IT has long been accepted that diabetes mellitus does not occur in the tropics and the Orient, or at least that its occurrence there is infrequent. Not much definite information is available on this point. That the situation, however, is either changing or better understood is indicated by an item in the India correspondence of the *British Medical Journal*, October 3, 1914 (p. 604), to the effect that the Madras Government, being impressed by the serious frequency of diabetes among the educated classes of that presidency, has appointed an investigator who is to give three years' study to the problems of causation and prevention. In regard to China, opinion is vague as to the prevalence of diabetes and the reasons for it.

To secure the data here presented, a questionnaire was sent to 150 representative western physicians scattered as evenly as possible through the eighteen provinces of China and Korea. Sixty-three replies were received, of which 28 reported no cases observed in periods of from eighteen months to thirty years' medical experience in China. Of the remaining 35 a number of case reports were so inexact and the diagnosis so lacking in adequate support as to preclude their consideration. Apparently reliable reports were obtained of 206 cases of diabetes mellitus,[1] of which the great

[1] Since preparing the above report, one case of diabetes mellitus has been treated in the Yale Hospital, making the total list 207 cases. This case died after two days in coma.

majority had been observed within the last five years. There were distributed by provinces as follows: Kwantung 53, Chekiang 34, Kiangsu 12, Fukien 12, Chihli 10, Hunan 8, Korea 8, Hupei 7, Formosa 6, Shansi 5, Shantung 4, Honan 3, Anhwei and Kiangsi 1 each; 42 not stated. Thus positive reports are in hand from 12 of the 18 provinces of China proper, as also from Korea and Formosa. The 28 negative reports were distributed by provinces as follows: 3 each from Korea, Chihli, Hupei, Hunan, Anhwei, and Manchuria: 2 each from Kwantung, Kiangsu and Szechuen: 1 from Honan and 3 not stated. No reports were received from Kwangsi, Yunnan, Kweichow, Shensi, and Kansu. There was a great diversity of opinion as to the relative incidence within China, observers in north or central China believing the disease more prevalent in southern and eastern districts, and *vice versa*.



Map showing distribution of diabetic cases by provinces.

Of the 164 cases which can be placed geographically, the largest number are in the coast provinces of Kwantung, Fukien, Chekiang and Kiangsu. These figures may not indicate a greater incidence in this area, but exact comparisons are nearly impossible to give. Various more or less indeterminate factors must be considered, such as the average temperament, social status and predisposition of the patients, the presence or absence of routine urine examinations, and the care and skill of the doctor in eliminating non-diabetic glycosuria. Until more exhaustive data are available, covering the entire 18 provinces, the question of relative incidence must be left in abeyance. Speculation from insufficient data is futile.

In none of the reported cases was definite hereditary predisposition noted. Of 136 cases where the sex was stated, 118 were in

males and 18 in females. This is not significant, as the great majority of all patients are males. So, too, with the age. Exactly two-thirds were between thirty and fifty years of age, but the majority of patients coming to hospitals and clinics are probably between these limits.

Age.	Cases.	Per cent.
Under 20	4	2.9
21 to 30	32	23.2
31 to 40	45	32.6
41 to 50	47	34.0
51 to 60	6	4.4
Over 61	4	2.9
	138	

Of 86 cases in which the occupation was stated, 48, or 55 per cent. were shopkeepers, tradesmen, or otherwise engaged in business; 20, or 23 per cent., were farmers. There were 8 housewives, 2 clerks, 1 tanner, 1 teacher, 1 coolie, 1 peddler, 1 soldier, and 3 men of means without occupation. In 26 cases the complaints for which medical relief was sought were noted as follows: 10 emaciation, 9 debility, 15 excessive thirst, 12 polyuria, 12 headache, 2 loss of weight, 4 pruritus, 3 peripheral nervous symptoms, 1 furunculosis, and 1 chronic cough. Closer examination of 64 cases showed a large percentage to be suffering from cataract and multiple abscesses. Dyspepsia, dysentery, and carbuncle were noted in three cases each, and pyemia, erysipelas, bronchopneumonia, and cryptogenic splenomegaly were found in one each. It is to be regretted that fuller information could not be elicited under these headings. It appears that patients presenting themselves with emaciation, polyuria, thirst, or headache, alone or in combination, especially if they have also multiple abscesses or cataract, and if no evident cause is found, should be examined for diabetes.

In 188 cases the predominant article of diet was named. In 130 rice was the staple and in 57 wheat, some of the latter adding millet, corn, beans, and sweet potatoes. Thus for the great majority, rice formed the main article of diet. It is out of the scope of this paper to do more than present the data collected. Fuller discussion is postponed to a later date.

Of 43 physicians who answered the question of relative prevalence, one considered diabetes more prevalent than in the west, 7 of equal frequency, and 35 considered it much less frequent. Among the reasons given for decreased frequency were the assumed fact that the Chinese as a race are hardier than western peoples, and that they resist nervous shocks better and have fewer of them. Little excitement and simple diet are adduced. One physician lays the decreased prevalence to the fact that the plain diet of rice and well-cooked vegetables with practically no sugars, is digested and absorbed so slowly as to prevent hyperglycemia. The

same correspondent continued that "the temperance habit (of the Chinese) in food and drink does not disturb the carbohydrate metabolism." It is at least an open question as to whether the Chinese as a race are as temperate in food and drink as this writer assumes. Fast eating without proper mastication, and periods of hunger terminated by most liberal and prodigious stuffing, together with a fairly constant consumption of alcohol, though rarely to the point of intoxication, are more characteristic of the race than is ordinarily supposed. It is more than possible that these factors may have a definite influence on the prevalence of arteriosclerosis and cardiac lesions, even in the first two decades of life. It is worth considering that the excessive lack of sugars in the average Chinese dietary, even in the presence of a maximum starch fraction, may well determine an increased tolerance for carbohydrates.

It is pointed out in a number of reports that diabetes in the Chinese is more prevalent in the better classes. This is probably true, and is confirmed by the distribution of the small series here reported, when it is remembered that the great bulk of the patients seen are from the lower classes. Dr. Lambert, of Kiukiang, whose observation and experience lend weight to his word, says: "I believe it has not until recently been a disease from which the Chinese have suffered much, but of late years, with the increased desire for foreign medical treatment evinced by Chinese of all classes, diabetes mellitus has been found to occur to quite an appreciable extent among the better classes." Lambert reported two cases of diabetes in the *British Medical Journal*, January 4, 1908, which were observed in Formosa. He also noted three cases of glycosuria in a series of 200 insurance examinations and one case of diabetes in 24,000 out-patients in Nanking, all drawn from the lower classes of society. He says, with reason: "Perhaps the idea that diabetes was an unknown disease among the Chinese arose from the fact that until comparatively recently foreign medical treatment was better appreciated by the coolie class than by the wealthier and more conservative merchants and officials, and because examination of wealthy natives for insurance was not so common in China ten years ago as it is today."

Dr. J. L. Maxwell, who found but two cases in 12,000 in-patients under accurate observation, concludes that he has no idea of the reason for decreased prevalence unless it be the absence of the bustle of the West. Even that he doubts, as he finds hysteria fairly common and both functional and organic nervous diseases not rare. Our experience at Changsha entirely confirms this statement. Dr. Maxwell, together with many, suggests the possible influence of racial tendencies and characteristics. It is useless at present to turn aside into this field, however attractive it appear, because it is as yet a field of speculation only. The peculiar racial psychology of the Chinese, based on controlled experimental data, is yet to be written.

Dr. Peill, of Tsangchow, Chihli, proposes the interesting thesis that "the only reason that occurs to me (for a prevalence less than in England) is the opposite of an unknown, *i. e.*, the opposite of that which makes it frequent among the Jews." Dr. J. P. Maxwell, who reports 11 cases from Yungchun, Fukien, considers diabetes more frequent than supposed. Dr. Park, from Soochow, Kiangsu, reports 8 cases in five years out of 3000 to 5000 persons seen annually. His practice is restricted to first-class patients. He says: "I used to think it infrequent, but the longer I stay in China the less am I inclined to make positive statements." This perhaps illustrates the probability already noted, that the rarity of diabetes in earlier and in many present reports may be in part explained by the fact that the great majority of patients coming to western hospitals are of the lower classes, where this disease is less apt to occur in any race.

Dr. Van Buskirk says that the disease is known to the Koreans, who call it the "sweet urine disease." He considers it rare, however. Another physician of thirty years' practice in Seoul had seen no case. Dr. Lewis, of Chenchow, Hunan, makes a comment which must always be borne in mind in a consideration of the prevalence of diabetes in China. He believes that the etiological factors ordinarily designated might be found as striking in China as in any prevalent district. Nevertheless, he considers diabetes very infrequent in south Hunan.

In regard to treatment, the prevailing opinion is that dietetic control with sufficient elimination of carbohydrates, brings the same results as in the West, but that dietetic control is usually impossible where rice forms the chief article of diet. Several favorable reports were received on the good results from substituting native oatmeal for rice. One diabetic dietary consisted of bran, meat, and goat's milk. One report described a case in a giant beggar, seven feet in height, who died in hospital. He was feeble-minded, and made his living by exhibiting his ability to eat five catties or seven pounds of bread at one sitting.

In view of the opinions at hand, representing the observation and experience of a large body of competent observers, the following conclusions seem justified:

1. In the predominant class of persons under medical observation in China, diabetes mellitus is less common than in the west.

2. It will, however, be found increasingly prevalent in China, as observation is extended more widely to the better classes of society.

3. The course of the disease and its response to treatment are parallel to similar conditions in the west.

4. No evidence is at hand indicating etiological factors different from those in the west. Theories accounting for a decreased prevalence may conceivably be refuted by widespread introduction of closer clinical examination.

REVIEWS

A Treatise on the Principles and Practice of Medicine. By Arthur R. Edwards, A.M., M.D., Professor of the Principles and Practice of Medicine and Clinical Medicine and Dean of the Faculty in the Northwestern University Medical School, Chicago; Attending Physician to Michael Reese Hospital. Third edition. Pp. 1022; 80 engravings and 23 plates. Philadelphia and New York: Lea & Febiger, 1916.

The recent advances in medicine is an aphorism oft repeated; the phase is, however, a true one and is well exemplified in the present edition of Edward's treatise. Since the presentation of the last edition of the work many advances have been made in medicine, and most of those relating to internal medicine have been incorporated by the author in his text. For example, he has found it necessary to write practically new sections on icteroanemia, the ductless glands, roentgen-ray findings, erythemia, sepsis, sporotrichosis, blastomy, trichinosis, hook-worm disease, pellagra, the cardiac arrhythmias, the numerous tropical diseases, anaphylaxis and a host of other disorders. The improvements in therapeusis are represented by the addition of Flexner's serum, tuberculin, the various forms of serum therapy, vaccines, salvarsan, as well as many less well-known drugs. In the present revision particularly praiseworthy are the amplifications in the chapters on tuberculosis and syphilis and the thorough discussion of such subjects as hypertension, constipation, drug addictions, and the neuralgias.

In the handling of each subject the general rule is followed to deal with under separate headings, the definition of the etiology, the symptoms, the diagnosis, the course and prognosis, and the treatment of the disease. This main plan is adhered to in all the disorders of more than minor importance while in the really important ones, or at least in the frequently seen diseases, there are separate headings devoted to the history, the pathology, the bacteriology, the differentiation, and the complications of the disease under discussion. In this way the author considers all the important phases of a disease, and yet is able to so condense the work that it makes a volume of good readable size which contains all the essentials in every particular.

The book can be highly praised, yet in several respects it would seem that certain criticisms might be made. The section on diseases

of the kidney seems to be one that is particularly deficient. The old pathological classification of renal disease is followed and no mention is made of the many functional tests that have been elaborated in the study of the nephropathies and upon which rational treatment depends. The discussion of the cardiac arrhythmias might well be enlarged, moreover, minor errors have crept into this section, as, for example, considering auricular flutter as synonymous with auricular fibrillation. The section devoted to gastric neuroses, a subject which one might say is a universal complaint, is extremely brief, while to typhoid fever, a rapidly decreasing disease, more space is allotted than to any other disease excepting tuberculosis.

These criticisms are more a question of opinion than a question of fact. With only an occasional error in facts here and there, the book is remarkably complete. It contains all the essentials; it is written in a lucid, clean-cut style, and it can be recommended as a most admirable text-book for the student or a reference book for the practitioner. J. H. M., JR.

DISEASES OF THE ARTERIES, INCLUDING ANGINA PECTORIS. By SIR CLIFFORD ALLBUTT, K.C.B., Regius Professor of Physics in the University of Cambridge. In two volumes. Pp. 534 and 559. London: Macmillan & Co., 1915.

THERE are certain medical treatises which stand out, for one reason or another, books which will always be of value and will have to be consulted and referred to in any discussion of the subject with which they deal. This work takes a place among these, and is also to be welcomed as the *magnum opus* of one who has long held a prominent place in medicine. It is not easy to write with a proper view of the wide problems of a subject when one is full of the knowledge of the details, and arterial disease has far-reaching effects. In this work there are both breadth of vision and attention to detail.

The author has written a preface which should not be missed: it is delightful reading. There will be no one to disagree with him that the labor put into these volumes is honest labor. To his hope "that the winter fruitage of an old tree, if a little withered, may not be unsound," we may say (altering the quotation slightly) that "age cannot wither, nor custom state, your infinite variety." Professor Allbutt has dealt with many subjects and has never failed to throw light on any one which he has discussed.

Diseases of the Arteries. The discussion of this occupies one volume and part of the other. As introductory to the subject proper there are chapters on the physics of the circulation and the viscosity of the blood. It is not difficult for the majority of us to

be beyond our depth before we know it when we venture in these waters. The chapter on the causes of arteriosclerosis extends to over 150 pages, and every possible factor is fully considered. The influence of mechanical and many toxic causes are discussed. Concerning the importance of alcohol in the etiology it is regarded as not a very important cause in itself, but a potent ally of any other poison. Of any influence of tobacco the conclusion is that we have little definite proof. The author insists on the distinction between arterial lesions with high pressure and without it, one which he has emphasized for many years. Special attention is paid to a form which the author terms senile or "decrescent arteriosclerosis." This he considers may be a family form in which the arteries are especially susceptible to toxic influences. It is not necessarily associated with high pressure.

Special interest belongs to the discussion of "hyperpiesis," a term which the author introduced to describe the cases in which high blood-pressure exists without renal disease. There is much difference of opinion as to the existence of such a condition and many deny that it occurs. But the evidence seems to support Professor Allbutt's contention, and we must agree that there are causes other than renal for persistent hypertension. In certain of these cases the increased blood-pressure is the cause and not the result of arteriosclerosis. The author insists on the importance of this, particularly with reference to treatment. In his opinion, if recognized early, the condition may be entirely recovered from, and in the majority of cases materially aided. Many disagree with this, but it is undoubtedly worthy of consideration, and those opposed to it should read this discussion of it with an open mind. Growing out of this view is the author's belief that cerebral hemorrhage is more often the result of high pressure than of disease of the arteries. Many would part company with him in this opinion.

In the discussion of symptoms it is pointed out that arteriosclerosis is to be regarded as a by-product of many diseases, and cannot be represented as a "specific symptomatic series." We are not able to say that its morbid anatomy is uniform. The chapter on arteriosclerosis and the kidneys discusses perhaps the most involved aspect of the subject. Chapters on the myocardium in arteriosclerosis, diagnosis and prognosis, and treatment conclude this section. There is so much detailed notice of many points that space forbids mention of them.

Angina Pectoris. The discussion of this is preceded by a chapter on aortitis, and very properly so in view of the author's views on angina, which he holds is the result of disease of the aorta, especially of the part just above the aortic valves. In the discussion of aortitis the etiological importance of certain of the acute infectious diseases is properly emphasized. The frequency of acute aortitis in rheumatic fever, for example, is usually not recognized; it is

common in children who have aortic endocarditis. In septicemia it will often be found if looked for. Excluding the cases due to acute infection the author lays great stress on syphilis. It is not very clear exactly what proportion of the cases he regards as luetic. The reviewer inclines to the belief that there are many cases in which syphilis does not play any part, in this regard chronic aortitis being decidedly different from aneurysm.

The various theories of the cause of the pain in angina are discussed in detail. The author holds firmly to the opinion that the first portion of the aorta is concerned and that the cause is tension of the outer coat. He does not regard disease of the coronary arteries as the cause of the essential phenomena of angina. Professor Allbutt has often insisted on the proper use of words, and finds excellent examples of the importance of this in his discussion of angina. He points out the many inaccurate statements as to the seat of pain which is described as being cardiac, precordial, or submammary when it is really substernal. The discussion of the character of the pain in angina is most illuminating. If we accept his views many of the difficulties connected with the disease disappears. Much interest attaches to the discussion of the simulations of angina; the term pseudo-angina is dismissed from consideration. These chapters are important in clearing away preconceived erroneous notions. In the discussion of the theories of angina, there are some hard knocks to fanciful ideas. Professor Allbutt certainly has made out a strong case for the aortic origin of the symptoms.

The chapter on the diagnosis of angina is a short one, as so much of the discussion of this aspect is gone over in considering the symptoms. The occurrence of pain from cardiac disorder is discussed and the distinction from angina emphasized. The diagnosis seems a more simple matter to the author than it does to the majority of the profession. There is a group for the diagnosis of which time is often necessary, but it is wise to regard them as serious until proved otherwise. The view of prognosis is perhaps more cheerful than that of the majority of writers.

It is not easy to review this book in proper proportion; a review could be written on each chapter. The literature has been extensively noted and clear accounts of cases given to illustrate special points. It is not necessary to speak of the author's style; it is too well known to require comment. There are few medical works which will give more pleasure in the reading than this, and the reviewer very heartily advises it, both for pleasure and profit. You will not agree with all the views expressed, but you will read with interest in any case. To write a treatise on two of the most obscure subjects in medicine and make it full of interest is somewhat of a task. We believe that this work will take a high place in the literature of medicine, and Professor Allbutt has placed the profession in his debt for this splendid clinical treatise. T. McC.

INFECTION AND IMMUNITY. A TEXT-BOOK OF IMMUNOLOGY AND SEROLOGY. FOR STUDENTS AND PRACTITIONERS. By CHARLES E. SIMON, B.A., M.D., Professor of Clinical Pathology and Experimental Medicine at the College of Physicians and Surgeons, Baltimore. Third edition. Pp. 351; 21 engravings and 12 plates. Philadelphia and New York: Lea & Febiger, 1915.

ORIGINALLY intended as an introduction to the study of infection and immunity and as a ground-work for more intensive and advanced study, this book of Dr. Simon's has been so enlarged that this the third edition might well serve as a text-book for students in a course of immunology. Moreover, the book will be found sufficiently large and amply comprehensive to serve the purpose of those practitioners who are interested in the practical side of the subject in its application to clinical problems. Lastly, as it has been written primarily for the general practitioner, the work will prove of inestimable value to the medical man who wishes a thorough grounding in the theoretical side of the subject even if he lacks the facilities to prepare the various laboratory tests as outlined by the author.

In the present edition the section on the Wassermann reaction has been greatly enlarged and so augmented that it will meet all requirements for the comprehension and performance of this important diagnostic aid. The Abderhalden test with complete technic have been incorporated in the text. The Schick test is carefully discussed, as is the production of antianaphylaxis. These are but a few of the many additions made in the new volume, but they will indicate, to a certain extent at least, how thorough and complete has been the revision.

Infection and Immunity can be most heartily recommended to the profession as a splendid reference and text-book on a subject which has only developed within a comparatively few years.

J. H. M., JR.

BONE-GRAFT SURGERY. By FRED. H. ALBEE, A.B., M.D., F.A.C.S., Professor of Orthopedic Surgery at the New York Post-Graduate Medical School and the University of Vermont, etc. Pp. 417; 332 illustrations. Philadelphia and London: W. B. Saunders Company, 1915.

DR. ALBEE has been for some years an indefatigable enthusiast in advocacy of what he terms "bone-graft surgery," and he has now succeeded in accumulating enough material to write a book about it. This is not meant to imply that the subject is not worthy of being recorded in book form, nor to incline anyone to underestimate the value of transplantation of bone in general, but merely to suggest that a little more conciseness of statement and a little

less repetition of steps in technic would have rendered the volume more readable as well as none too small. As it stands now there is too much "padding;" the book has not received the editing which is its due.

The volume opens with chapters on fundamental principles and on the author's electric-motor outfit. There follow chapters devoted to the use of bone transplants in spinal affections, in fractures, in fixation of the larger joints, and in various miscellaneous affections such as club-foot, infantile paralysis, and after excision of the diaphyses of the long bones for osteomyelitis or tumors.

Some people have objected to Dr. Albee claiming so much credit for his work. They point out that there were heroes before Agamemnon, only they didn't blow so loud a trumpet. Now very likely this is true; but the fact remains that the man who popularizes, not to say exploits, a method of practice deserves very great credit for his exploitation, if for nothing else. This was the case of Gouley with the tunnelled catheter, of Freyer with suprapubic and of Young with perineal prostatectomy, and of Murphy with arthroplasty. It is true, of course, that many surgeons before Albee transplanted bone, and that his electric-motor outfit is only a modification of the Hartley-Kenyon motor, and that these latter gentlemen did not themselves invent electricity; and it is true that Hibbs was as early if not an earlier advocate of bone transplantation for spinal caries, and that even Albee's favorite "inlay method" was described in print in a leading article by Buchanan before (as much as one month before) Albee, in the closing paragraphs of a long paper, made rather incidental reference to the same method as used by himself. But if Buchanan can afford to ignore any claims for priority, why should anyone else object? Albee has established the method on a firm basis, and by his large experience in all varieties of bone transplantation has become a recognized authority. Wherefore, it is entirely fitting that he should publish a book on the subject.

What may perhaps be regarded as a just objection is that he permits his enthusiasm for bone transplantation to warp his judgment respecting the value of other and simpler surgical procedures. Like many other writers in recent years he blames all the bad results (and there are a great many) following the use of metal plates on the metal plates instead of on their inefficient use or on the lack of judgment and knowledge in the surgeon who employed them. There can be no question in the mind of anyone with sufficient experience in such matters that *metal plates are perfectly satisfactory for the treatment of recent fractures when properly applied.* Another criticism which we believe is just, is that Dr. Albee often strains at a gnat and finds he has swallowed a camel. Opening the volume at random just now, an instance of this greets the eye (p. 335): "Astragalus used as transplant to arthrodese ankle"

is heralded as an epoch-making contribution to surgery; what was actually done was to produce ankylosis in the ankle and sub-astragalar joints by removing the astragalus and reimplanting it after properly denuding its articular surfaces and those of the neighboring bones. Now such an operation is currently done by orthopedic surgeons without disturbing the astragalus from its bed at all: the result is quite the same, and the procedure is much simpler.

But allowing for such shortcomings, due to acquired mental astigmatism, and ignoring carping criticisms, the volume remains an admirable record of work well done—an example for others to emulate. A. P. C. A.

GYNECOLOGICAL OPERATIONS, INCLUDING NON-OPERATIVE TREATMENT AND MINOR GYNECOLOGY. By HENRI HARTMANN, SC.D., Professor of the Faculty of Medicine, Paris; Surgeon to the Laennec Hospital, Paris; Member of the Society of Obstetrics and Gynecology; Member of the Society of Surgery; Member of the International Society of Surgery; Former President of the French Congress of Gynecology; Honorary Fellow, Clinical Society of London; Honorary Fellow, Royal College of Surgeons, Ireland. Translated by DOUGLAS W. SIBBALD, M.B., CH.B. (Edin.), formerly Physician to the British Hospital, Levallois-Perrot, Paris. 422 illustrations. Philadelphia: P. Blakiston's Son & Co., 1913.

THERE can be no doubt that a translated work often loses much of its value in the process of translation, since the literary style of the author is not easily expressed in a foreign tongue. In the work under consideration the translation has been done in a very mediocre manner, perhaps on account of the effort of the translator to stick too closely to the original text. The English construction is very poor indeed, errors in spelling are common, and there are many evidences of carelessness in proofreading. The illustrations do not approach the standard which we have become accustomed to expect in works of this character, most of them being made from wood-cuts. A redeeming feature of the book is the fact that the type is large and very easily read. Although, as the title indicates, the book is intended to be a dissertation on the operative side of gynecology, with no attention given to symptomatology, considerable space is devoted to non-operative therapy and minor gynecology, the subject of electrotherapy being well handled by Zimmern. Many of the procedures advocated by Hartmann as methods of choice are seldom employed in this country today, such as dilatation of the uterus by means of laminaria tents and morcellement in the treatment of uterine fibroids. Curettage is discussed at length,

the author favoring its use in septic abortions. The best articles in the book are those devoted to hysterectomy, each of the more prominent methods of performing this operation is considered in turn, followed by the various modifications of these methods, concluding with the indications, contra-indications, and complications in each case. This part of the work, especially the chapter on vaginal hysterectomy, is well illustrated. The disorders of menstruation are mentioned in a very superficial way; chapters on the urinary organs, especially the treatment of the various urinary fistulæ, are most satisfactory. The book contains much information, but needs considerable polish and revision. F. E. K.

DIARRHEAL, INFLAMMATORY, OBSTRUCTIVE AND PARASITIC DISEASES OF THE GASTRO-INTESTINAL TRACT. By SAMUEL GOODWIN GANT, M.D., LL.D., Professor of Diseases of the Colon, Sigmoid Flexure, Rectum and Anus at the New York Post-Graduate Medical School and Hospital. Pp. 604; 181 illustrations. Philadelphia and London: W. B. Saunders Company, 1915.

As a corollary to his recently published work on constipation, Dr. Gant has brought out a book on the adverse condition, diarrhea, which follows very much on the same lines the plan pursued in the original work. The book is an extremely careful compilation of all the various types of primary diarrhea, that is, the diarrheas that occur as a result of intestinal diseases and of the manifestations of diarrhea that occur in conditions which have no direct relationship to the intestines. As would be expected in a book of this type, the major part of the text is devoted to treatment which includes not only drug therapy, but also mechanical treatment, and treatment by operative procedure; to this last method of treatment incidentally some fifty pages are devoted. However, the other phases of diarrheal diseases are not neglected by any means, so that the work will prove a splendid reference book upon a protean subject. J. H. M., JR.

THE MEDICAL CLINICS OF CHICAGO. Vol. I, No. 4, January, 1916. Pp. 221; 23 illustrations. Philadelphia and London: W. B. Saunders Company, 1916.

THE fourth number of the *Chicago Medical Clinics* opens with an excellent discussion of epidemic cerebrospinal meningitis by Dr.

Tice. In the clinics devoted to this subject he details the technic of lumbar puncture and the examination of the cerebrospinal fluid in a most thorough and careful manner. The next two clinics by this author deal with Pick's cirrhosis and with acute endocarditis complicated by meningitis.

Dr. Hamburger follows with an equally good exposition on primary carcinoma of the liver, the pathological side of which is discussed by Dr. Kuh.

Dr. Weaver then gives a most pertinent talk on the Schick reaction.

In Dr. Mix's presentation of a case of upper lobe pneumonia he lays particular stress, as prognostic signs, upon accentuation of the pulmonic second sound, the relation between pulse-rate and blood-pressure, and the occurrence of arrhythmia. The first of these is undoubtedly of some prognostic value, the second is an extremely doubtful prognostic sign. Opinion varys considerably concerning the value of Gibson's rule, but certainly most clinicians doubt its prognostic worth. As to the question of arrhythmia, the type or types of arrhythmia which lead to fatal result in pneumonia are not specified. Hundreds of children recover from pneumonia who have had well-marked sinus arrhythmias during the course of the disease.

Dr. Hamill's neurological clinics, which are next in order, are carefully prepared and interestingly presented.

Cases of malaria, hemorrhagic pleurisy and trichinosis are shown by Dr. Williamson in his clinic.

Dr. Preble's bedside clinic is spoiled for the reader by entirely too much questioning and subsequent answering by the "visitor."

Dr. Goodkind's contributions are a case of pneumococcic cerebrospinal meningitis and a case which is called aplastic anemia but which lacks many of the characteristics of this type of anemia. The author evidently is not sure of his diagnosis in this case because he says in the text that he is justified in saying that the diagnosis is pernicious anemia of the idiopathic type.

La grippe in infants, a clinic by Dr. Alt, which is well worth reading, concludes the present number of the *Clinics*.

J. H. M., Jr.

Manual of Embryology. By A. Melville Paterson, Professor of Anatomy in the University of Liverpool. Pp. 391; 304 figures. New York: Oxford University Press, American Branch, 1915.

In this printed version of his lectures on embryology the author has succeeded in reducing to a compact and easily understandable form the important facts of his subject. It will be useful to all who wish a definite answer to questions concerning the prenatal history of the organs and systems. The author's style is terse and lucid,

and the book is illustrated with numerous diagrammatic figures, which help much to elucidate the text. Many of these are adapted from previous works, but the author has also evolved a number of original ones. Everywhere the history of the organs leads up to the fully formed condition, and for that reason will appeal especially to those whose acquaintance is with gross rather than with microscopic anatomy. The keynote of the book is evidently clearness and simplicity. This, of course, has the disadvantage of preventing discussion of controversial points, and makes some of the statements unduly hard and fast. However, for the beginner or for the man whose laboratory instruction in the subject has only been elementary, this form of manual will be very welcome. W. H. F. A.

PRACTICAL CYSTOSCOPY AND THE DIAGNOSIS OF SURGICAL DISEASES OF THE KIDNEYS AND URINARY BLADDER. By PAUL M. PILCHER, M.D., Consulting Surgeon to the Eastern Long Island Hospital. Second edition, thoroughly revised and enlarged. Pp. 504; 299 illustrations. Philadelphia and London: W. B. Saunders Company, 1915.

A DETAILED review of this second edition of Pilcher's book might well be compared to the introduction of old friends who had merely modified their garb to suit a more modern style. Four years have elapsed since the first edition was published, and during this time the book has become well and favorably known. The general arrangement of the first edition remains unchanged, one or two new chapters have been added, and others amplified or altered to embody the more recent developments. The chapter on pyelography is new, and is a concise statement of facts concerning its indications, value, technic, and dangers. At the conclusion of this chapter are pyelograms, furnished by Braasch, demonstrating the various types of renal lesions. The chapters on diseases of the prostate and functional diagnosis have been entirely rewritten; under the latter heading only the more generally used and accepted tests are described in full, the others being mentioned but passed by with a few words. We do not agree with the author's general statement that the phthalein test "is interfered with by parenchymatous nephritis, but does not seem to be influenced by chronic interstitial nephritis." The latter portion of the book details the therapeutic uses of the cystoscope and the application of the high-frequency current in various diseases of the bladder and ureter.

This book while intended primarily for the specialist must appeal to the man in general work as well, taking up as it does the clinical history and diagnosis of the various diseases of the urinary system.

It can be recommended as a practical, up-to-date presentation of the entire subject. F. E. K.

HANDBUCH DER TUBERKULOSE. Edited by PROF. DR. L. BRAUER, Hamburg-Eppendorf, Allg. Krankenhaus, DR. G. SCHRÖDER, Dirigierender Aerzt der neuen Heilanstalt f. Lungenkranke zu Schömberg, O. A. NEUENBÜRG und DR. F. BLUMENFELD, Aerzt f. Nasen, Hals- und Lungenkrankheiten in Wiesbaden. Vol. I (in two sections). Pp. 792; 88 illustrations; 18 plates. Leipzig: Johann Ambrosius Barth.

THE first volume of the five comprising this system is divided into two sections, the first dealing with the history, pathology, and epidemiology of the disease, and the second covering diagnosis.

The articles are so comprehensive and cover the subject so thoroughly and in such detail that it is very difficult to select any articles for special consideration. The pathological anatomy of tuberculosis is covered by Tendeloo in an article containing a wealth of information illustrated by carefully selected plates. This subject is so important for a thorough understanding of the disease that it deserves special mention, although it is unfortunate that this chapter is not brought into closer relation to the chapter dealing with the study of the clinical types (Meissen, second section), as it would have materially added to the value of each.

Much's article on the tubercle bacillus is largely devoted to a presentation of the writer's views in regard to the significance of the granular rods and granules which he has described, a long list being given of the writers who have supported his views and a few whose investigations failed to confirm his findings.

The various theories held in relation to infection, especially infection in childhood, have been very carefully reviewed in the article by Römer. Much's article on immunity, Gottstein's on epidemiology, and Dammann's on the prevention of tuberculosis in animals add considerable value to this volume as a work of reference.

The second section will probably have a more general interest among clinicians, especially the article on diagnosis by Brecke which is exceptionally good.

In view of the fact that the study of the temperature as an aid in diagnosis is usually ignored both by writers and clinicians, it is worthy of note that the portion of the article dealing with this subject is very complete, containing a review of numerous studies and several observations by the writer. The portion dealing with the history of the patient seems to be unnecessarily brief.

The articles by Ritter, on the clinical significance of the tuberculin reactions, and by Lorey, on the use of the roentgen-rays, are very thorough and exhaustive presentations of the respective subjects, although the claims made for the roentgen-rays, as is so frequently the case, seem rather extravagant.

Thoracoscopy and laparoscopy as described by Jacobaeus, as a

means of diagnosis, will probably never be very widely adopted by clinicians in this country.

This system, if the succeeding volumes maintain the standard set up by this volume, will be of value to the general clinician mainly as a work of reference; while those especially interested in the study of this disease will find it invaluable. It is especially unfortunate that in a work containing such a wealth of information of an accurate and scientific nature the material should be so inaccessible on account of the lack of systematic grouping and arrangement of the text, a failing so common to works of German origin. The references alone make the work valuable, although the references to English and American writers are conspicuous by their rarity. F. A. C.

PHARMACOLOGY, CLINICAL AND EXPERIMENTAL. A GROUNDWORK OF MEDICAL TREATMENT. By H. H. MEYER and R. GOTTLIEB, Professors of Pharmacology at Vienna and Heidelberg. Translated by J. T. HALSEY, Professor of Pharmacology, Therapeutics, and Clinical Medicine at Tulane University. Pp. 604; 64 illustrations. Philadelphia and London: J. B. Lippincott & Co.

CONSIDERING pharmacology as the "reaction of living organisms to various chemical agents," the authors have made an able presentation of scientific drug therapy, with frequent references to physiology and the seat and cause of the pathological conditions involved. As its name implies their book is more than a text-book of therapeutics in that it considers the pharmacological action of certain drugs not used in medicine (*e. g.*, curare, the first drug discussed). On the other hand it does not attempt to include remedies other than drugs, such as massage, electricity, baths and so forth.

A fundamental division of all drugs is made into two classes: organotropic (drugs influencing organs and their functions) and etiotropic (those acting on the causative agents of disease). The table of contents is divided into eighteen chapters, the first fourteen being devoted to the pharmacology of the several organs; the last four to the pharmacology of heat regulation, of inflammation, etiotropic pharmacological agents, and factors influencing pharmacological reactions. Drugs are not considered individually, but under each organ that they affect pharmacologically. A comprehensive list of original sources is given at the end of each chapter. All doses are given in the metric system only.

Numerous additional facts and opinions are inserted in brackets by the translator. These so frequently disagree with the authors, and sometimes supply such serious omissions, that an original

work by the translator would be awaited with interest. For example, the important effect of digitalis on the conductivity of the heart, so well demonstrated by Mackenzie, Cushny, and Lewis, is briefly dealt with between brackets on page 266.

The subject matter appears on the whole to be accurately and adequately given. Many of the newer synthetic compounds are given, with cautious estimation as to their probable worth. Among the cathartics the phthaleins are emphasized, including the phenoltetrachlorphthalein as advocated by Abel and Rowntree. One is surprised, however, to find no mention of agar-agar or paraffin oil among the laxatives.

The book is well bound and well printed on good paper, and misprints are few. A dangerous slip occurs on page 38, where 0.02 gm. of pantopon is said to correspond to 0.1 gm. of morphin instead of 0.01 gm. morphin. E. B. K.

Selected Addresses. By James Tyson, M.D., LL.D., Professor of Medicine Emeritus, University of Pennsylvania. Pp. 366. Philadelphia: P. Blakiston's Son & Co.

The fifteen addresses collected in this volume were made by Dr. Tyson on various occasions begween the years 1884 and 1910. There are also included three articles descriptive of European spas.

The addresses cover a wide range of subjects: education, biography, travel, and medicine. Noteworthy are those on college education, which contain much that is descriptive of Dr. Tyson's undergraduate life at Haverford College.

The presidential address at the dedication of the new hall at the College of Physicians of Philadelphia is of particular interest by reason of the many references to the early medical history of this country.

The work is carefully edited, and the addresses from first to last are written in the scholarly manner which has always characterized Dr. Tyson's literary contributions.

This volume should be of particular interest to his former pupils, of whom some six thousand have been graduated during his connection with the Medical School of the University of Pennsylvania.

R. G. T.

PROGRESS
OF
MEDICAL SCIENCE

MEDICINE

UNDER THE CHARGE OF

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Indicanemia.—Haas (*München. med. Wchnschr.*, August 3, 1915) calls attention to the diagnostic importance of an increase in indican in the serum. It was first recognized by Obermayer and Popper, who found that indican was practically never present in the serum except in uremia, in which condition it is always found in greater or less amounts. The author states that it is often present not only in uremia, but also in chronic nephritis of all types and it is not confined to those cases showing a nitrogen retention in the blood. It is a better sign of renal insufficiency than increased rest nitrogen.

Concerning Septic Meningococcus Infection.—Bittorf (*Deutsch. med. Wchnschr.*, 1915, No. 37) cites four instances of meningococcus infection with all the characteristics of a general septicemia and with the recovery of the meningococcus from the blood in one case. He regards the disease as a general septic infection in which the meningitis constitutes only one feature of the general picture and is due to organisms brought by the blood stream rather than the usual view that it is secondary to direct invasion from the naso-pharynx. The following points are offered in support of his view that the meningitis is only a part of the picture of a general septicemia: (*a*) The presence of a typical septic embolic eruption with the early round or oval dark-blue spots in the skin which later become definitely infiltrated and in some instances go on to pustule formation. (*b*) Numerous septic metastatic symptoms, oftenest multiple, swollen and painful joints, endocarditis and in one case hemorrhagic nephritis. (*c*) The appearance of an acute splenic tumor in three out of the four cases. (*d*) Septic temperature with chills and jaundice. (*e*) The recovery of the organism from

from the blood in one case. In all cases the meningococcus was recovered from the spinal fluid and the possibility of a myeloid infection to account for the general septic picture presented by these patients was excluded from the onset. The appearance of meningitis later in the course of the disease has only the same significance as the joint and skin manifestations. This is further supported by the finding of only a very few organisms and a moderate cellular increase in the early lumbar puncture, but with a marked increase in both elements is the fluid withdrawn later. These observations agree with those of Ostermann (1906 and 1907) who considers the disease a hematogenous infection with the portal of entry in the naso-pharynx while the tonsils seemed the more likely source in these cases. The cultivation of the organism from the blood has also been previously reported by Westenhoeffer, Salomon and Martini, and Rhode. All the cases reported recovered from the meningitis (one died suddenly during convalescence) with the use of frequent lumbar puncture, large doses of salicylic acid and urotropin when the meningitic symptoms started. Antimeningitis serum was not used in any case.

The Use of Brain Extracts as an Antigen in the Wassermann Reaction.—Maruyama (*Jahrb. f. Psych. u. Neur.*, 1914) has taken up the question as to whether extracts of paretic brains would serve to give specific complement fixation in the Wassermann reaction. He made various types of extracts, such as plain alcohol, alcohol ether and acetone, and tested all of these antigens against a rather wide variety of cases, both luetic and otherwise. Though the series is but small, and in the author's opinion warrants no positive conclusions, he has felt justified in arriving at the following facts: (1) Alcohol and alcohol ether extracts of brain substance are of no use as antigens in the Wassermann reaction. (2) Acetone extracts, however, seem to give results of a very valuable and specific nature in the Wassermann reaction. The question remains open as to whether these antigens contain an unusual amount of cholesterin or not, but he is rather inclined to believe that they do. In only one case did he note a negative result with the acetone extract in a case where the blood serum gave a positive reaction when tested with the usual type of antigens. A number of suggestive leads are opened up by this study which may be of service in throwing more light upon the nature and specificity of this much used reaction.

Concerning the Function of the Spleen.—After reviewing the various ancient theories and more recent knowledge of the function and importance of the spleen, Hirschfeld (*Deutsch. med. Wchnschr.*, 1915, Nos. 37 and 38) feels that the same relation between the organs of blood building and blood destruction exists as between the various glands of internal secretion. The profound disturbance of erythropoiesis in the bone marrow following the loss of function can scarcely be explained except on the basis of an interacting internal secretion. The present state of our knowledge concerning the function of the spleen, he summarizes as follows: (1) The human spleen is a blood building organ. In the normal person it is not concerned in the building of red cells but is a factor in the production of lymphocytes and

large mononuclears. Under certain pathological conditions, such as leukemias, anemias and infectious diseases, the spleen can take on the function of building all the blood elements. (2) Normally the red cells are destroyed in the spleen and the hemoglobin starts there the subsequent change into bilirubin in the liver. It thus plays a helpful part in conjunction with the liver in hemoglobin metabolism. (3) It is one of the organs concerned in the iron metabolism which substance it can store and thus help maintain the hemoglobin content of the body at its normal element. (4) It acts as a lymph gland and is not only a burial ground for the red cells but for the white cells and any foreign substance circulating in the blood. The latter accounts for the well-known changes in the spleen seen in infectious diseases. It is also one of the places for the building of immune bodies against bacteria. (5) It is a regulator of the erythroplastic function of the bone marrow. Splenectomized animals and men show in the circulation red cells containing Howell-Jolly bodies, a symptom of disturbed erythroblastic regeneration. In occasional cases this disturbance leads to a true polycythemia. (6) None of the functions of the spleen are vital, as they are often very quickly taken over by other organs and the patient lives normally after extirpation of the spleen, without any disturbance in health. (7) For most individuals the spleen is unnecessary. Yet there are people in whom splenectomy leads to a definite and severe disturbance with polycythemia.

SURGERY

UNDER THE CHARGE OF

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Clinical Results of 1000 Intravenous Injections of Galyl.—SPENCE (*Lancet*, December 11, 1915, p. 1292) says that no systematic study has been undertaken of the effect of the injections on the Wassermann reaction or of certain other questions chiefly of academic interest. Galyl, or tetraoxydiphosphaminodiarsenobenzene, is one of several synthetic substitutes for salvarsan which have recently appeared on the market. A comparison of the full doses advised for galyl and for salvarsan and neosalvarsan indicates that galyl as administered actually contains only about half as much arsenic as the others, a point of passing interest in view of the recent suggestion that the activity of these agents may be not wholly dependent on the arsenic moiety.

The preparation is supplied in five different strengths—viz., 0.4 gm., stated to be the full dose and to correspond to 0.9 neosalvarsan; three smaller, 0.25, 0.30, and 0.35 gm.; and a larger, 5 gm. It is left to be inferred that the large dose is to be used only very occasionally, or in other words that it is a specially powerful one. It is used in precisely the same way as neosalvarsan, either in concentrated solution in the 10 c.c. piston syringe, or more dilute in the familiar burette apparatus, preceded and followed by a little distilled water. A perusal of the histories will at once suggest that in clearing up symptoms the new preparation differs little if at all from its older rival. Second only in importance to effectiveness in clearing up symptoms is the question of toxicity. More than 150 deaths are credited to salvarsan, to say nothing of innumerable accidents and severe reactions. So far as Spence is aware no fatality has as yet been reported with galyl. Neither has there been reported any undoubted case of arsenical intoxication, but in this connection it is to be remembered that the remedy is new and its literature very meagre. An interesting complication met with twice was a reaction resembling serum sickness, large urticarial wheals which itched intensely, appearing a few hours after administration and remaining two or three days. Seven cases exhibited a pseudo-Herxheimer reaction, all the symptoms seeming to be temporarily aggravated, the throat more congested and painful, and rash and malaise accentuated. It is, however, but fair to say that any one of the reactions and symptom just enumerated is at least as frequent with salvarsan and neosalvarsan, and none is typical of arsenical poisoning, very probably being due, as the studies of Yakimoff and others indicate, to the release of various bacterial endotoxins.

Intestinal Stasis.—Ochsner (*Surg., Gynec. and Obst.*, 1916, xxii, 44) says that during the year ending December 31, 1914, operation to correct intestinal stasis was performed on 36 cases, with 32 recoveries and four deaths. These cases represent less than 10 per cent. of all the cases examined during this period, and treated for intestinal stasis, and still the number operated upon contains some cases which should not have been treated surgically, as has been shown by their subsequent condition. This is true especially of a few neurotics who undoubtedly suffered as a result of intestinal stasis with marked auto-intoxication due to absorption of products of decomposition. All of these neurotic cases showed marked improvement after operation. In a certain proportion, however, this seemed to last only until some unusual or unexpected circumstance upset the regularity of the digestive process, when they seemed to glory in the consciousness that they were again enjoying a type of ill health which they could attribute to the abnormal condition of the intestinal tract. In some of these cases a second or third operation was performed, but only rarely with the result of obtaining permanent relief. There can be no doubt but that the surgical relief of demonstrable mechanical obstruction causing stasis will in many instances give complete or almost complete relief, the result depending upon the degree to which the stasis is due to a removable mechanical obstruction. In a very large proportion of the cases, however, the cause is not single and for this reason one should look for only partial relief following surgical treatment. Only a very small

percentage of patients suffering from intestinal stasis will need to be considered from the surgical standpoint. This applies to those in whom there is a possibility of overcoming intestinal stasis by removing the cause of the obstruction existing in the form of tumors pressing upon the intestine, the correction of uterine displacement compressing the rectum, obstruction due to bands of adhesions, or marked kinking of intestines, of strictures due to cicatricial contraction following ulcer of the intestine, of annular carcinoma of the intestines and occasionally of large papillomata, fibromata, or lipomata projecting into the intestine. If for any reason the obstruction cannot be relieved in this way then short circuiting must be resorted to, and if the colon is dilated with its muscular walls so seriously impaired that the power of contraction has been permanently destroyed, then at least the cecum and ascending colon should be removed; and in severe cases it may become necessary to remove the entire colon down to the sigmoid flexure.

The Abdominal Skin-flap in Radical Amputation of the Breast.—Elsberg (*Ann. Surg.*, 1915, lxii, 678) says that the abdominal skin-flap is necessary only in those cases in which the defect left after radical amputation of the breast cannot be closed by any of the methods ordinarily in use; it is useful after extensive removal of the skin and makes skin grafting unnecessary. The skin of the abdominal wall is generally very lax and receives abundant blood supply from branches of the arteries which run through the wall of the abdomen from behind forward. If the base of the skin-flap is not too small the tissues will be well supplied with blood, and there is no danger of sloughing or of marginal skin necrosis. After the flap has been raised from the fascia and slid upward into a new position, there is no difficulty in closing the defect in the abdominal wall with interrupted sutures. It is surprising how much skin can be used for the flap and sufficient remain to close up the defect in the abdominal parieties without much tension. The size and shape of the abdominal skin-flap will depend upon the size and shape of the raw surface on the chest wall.

Operation for Relapsed Club-foot.—Cook (*Amer. Jour. Orthop. Surg.*, 1916, xiv, 9) presents the following description of the operation which, in his personal experience, has proved the most useful for relapsed club-foot in cases that have resisted the ordinary methods of treatment. There is no age limit, no preliminary treatment and no after-treatment beyond the time required for the healing of the wound. (1) If necessary subcutaneously divide the fascia on the inner side of the foot and also the heel cord; then bring the foot into as good position as possible, using nothing but the hands and being careful not to bruise the tissues. (2) Make an incision through the skin and superficial fascia just in front of the external malleolus on the outer side of the foot. The skin incision should be perpendicular from the bottom of the foot to just above the bend of the ankle. (3) With an osteotome remove a large wedge of bone; make the first incision far back, just in front of the fibula. Pay no attention to the periosteum or peronei tendons. Cut the bones completely across and remove everything. Be sure to make the wedge large enough. The foot can now be brought without force into excellent position, and by giving the anterior part

of the foot a quarter turn, its outer border can be elevated. It is vitally essential to the success of the operation that the outer border of the foot be elevated. In order to do this the tarsus is cut completely across to enable the operator to give the anterior foot the quarter turn which elevates the outer border. Cook uses a light retention splint in preference to a plaster cast. Young children often walk at the end of two weeks. He has been doing the operation for the past twenty years, and during the last three and a half years exactly as described above, operating on 30 patients in all, in some cases on both feet. Three of the feet have relapsed and a secondary operation was necessary, probably, because a sufficiently large wedge of bone was not removed in the first place. All of the patients are cured and all have practically perfect functional use of their feet.

Technic of Thyroidectomy.—QUERVAIN (*Deutsch. Ztschr. f. Chir.*, 1916, cxxxiv, 475) says that he has worked up a technic for goitre operations which he has employed for five years, and the chief points of which are as follows: He ligates the arteries, especially the inferior thyroid, when it does the most good, before the gland is luxated. The ligation of the usually well developed inferior thyroid should be extrafascial, outside of the gland fascia derived from the cervical fascia, because one can avoid most surely here the structures inside of the gland fascia, the recurrent laryngeal nerve and the parathyroids, as well as the capsule veins. The best approach is afforded by the sternohyoid space, *i. e.*, the space in which the small thyroid gland muscles are found, and best to the outer side of these muscles but inside of the strong fascia covering them. Whether the goitre tissue is to be removed by eneucleation or resection will depend upon the form of the goitre and has no special importance. The isthmus is to be divided and partly or wholly removed, only when it is itself diseased. In all other cases its division is superfluous and a disadvantage, because the collateral vessels at its upper and lower borders are damaged. These to some extent provide nourishment to the stump after the ligation of both arteries of one side. Whenever possible, as much of the gland is to be left on each side as corresponds to the normal gland lobe. The posterior surface of the lobe and, therefore, the region of the recurrent laryngeal nerve and parathyroids, will remain untouched and neither of them will be exposed. The parenchymatous hemorrhage will be controlled by a fine catgut suture which unites the two edges of the defect remaining in the gland.

Studies on the Localization of Cerebellar Tumors.—GREY (*Ann. Surg.*, 1916, lxiii, 129) reports a study from the surgical clinic of the Peter Bent Brigham Hospital, Harvard Medical School, with particular reference to the position of the head and suboccipital discomforts. Of 58 certified cases of cerebellar and extracerebellar tumor an unusual attitude of the head—tilted so that the ear approximated one shoulder, or both—was found in 23. In the majority of these the change in position was slight. Of 43 certified cases of tumor lying anterior to the cerebellum only 3 showed any tilt or rotation of the head. The unusual attitude in these 3 cases was scarcely noticeable. About 40 per cent., then, of the cases with cerebellar tumor showed some change in the

position of the head while only 7 per cent. of the cases with tumors anterior to the cerebellum showed any unusual attitude. These findings indicate that a tilt or rotation of the head in a patient with symptoms pointing toward an intracranial tumor is suggestive of a subtentorial newgrowth. Such an attitude, however, has no additional significance in localizing the lesion in one or other side of the posterior cranial fossa. Backward retraction of the head was a feature in 8 of the 60 cases of cerebellar and extracerebellar tumor. Typical opisthotonos attacks appeared in 2 of these. A similar position was noted in none of the cases with tumors lying anterior to the cerebellum. Backward retraction of the head, then, is characteristic of newgrowths situated below the tentorium. While atrophy or osteoporosis of the subjacent bone may occasion occipital tenderness in certain cases, comparisons of the clinical and operative findings in this series of cases have shown no consistent relations to exist between the two. Of 59 certified cases of cerebellar and extracerebellar tumor some form of suboccipital discomfort was present in 44, about 75 per cent. Tenderness in the occiput was found in 21, 36 per cent. Suboccipital headache or pain was complained of in 33, 56 per cent. There was more or less soreness or stiffness of the neck muscles in 18, nearly 31 per cent. Accordingly, headache or pain is the most frequent of the suboccipital discomforts. Of 43 certified cases with tumors lying anterior to the cerebellum some degree of suboccipital discomfort was found in 14, approximately 33 per cent. Suboccipital tenderness was present in 8, nearly 19 per cent. Suboccipital headache or pain appeared in 10, about 23 per cent. Soreness or stiffness of the neck muscles was complained of in 6, 14 per cent. As a rule the occipital discomforts were much less intense in the cases with tumors situated anterior to the cerebellum than in those with subtentorial newgrowths. Taking the series as a whole, no consistent relation has been found between the part of the posterior cranial fossa occupied by the tumor and the site of the discomfort. When unilateral suboccipital discomfort is present, however, it is slightly suggestive of the site of the newgrowth. These findings indicate that suboccipital discomforts are present more than twice as often in patients with subtentorial newgrowths as in those with tumors situated elsewhere in the brain; and, though they afford only slight assistance in localizing the lesion in one or the other side of the posterior cranial fossa, they nevertheless rank with asynergy (limb ataxia, staggering gait, etc.) as the most important indications of a subtentorial localization of intracranial newgrowths.

Fractures of the Spine Without Paraplegia.—Hartwell (*Am. Jour. Orthop. Surg.*, 1916, xiv, 82) says that during a six months' service in the male surgical division of the out-patient department of the Massachusetts General Hospital—from February 1, 1915, to August 1, 1915—ten patients sought relief for pain in the back who were found to have a fracture of the spine. And during this same period one patient with spinal fracture was treated in the orthopedic division. These patients were all ambulatory, and attempts to elicit a history of symptoms that might indicate cord compression were vain. Not one had at any time had any motor or sensory paralysis or sphincteric disturbance. In a review of 133 cases of spinal fracture treated at the Massachusetts

General Hospital between January 1, 1900 and December 31, 1914, 106 patients were found to have applied for treatment within forty-eight hours of the receipt of the injury. Fifteen per cent. of patients with fracture in the cervical region, 32.4 per cent. of patients with fracture in the dorsal region, and 48.3 per cent. of patients with fracture in the lumbar spine, presented no signs of damage to the spinal cord. A study of the subject is summarized as follows: Fractures of the vertebral bodies and lumbar transverse processes are not infrequent injuries but are generally overlooked. These fractures cause steady pain in the back but need not be accompanied by any neurological symptoms. Localized tenderness over the spinous processes should always suggest the possibility of fracture of the spine, and if in addition to localized tenderness there is a disalignment of the spinous processes, the diagnosis is almost certain. The kyphos caused by compression fractures may readily be mistaken for the deformity of a bad posture. Treatment should consist of prolonged fixation of the spine in extension by plaster jackets or permanent splinting of the spine by a bone grafting operation.

The Physiological Treatment of Bullet and Gunshot Wounds of the Peripheral Nerve Trunks.—LYLE (*Surg., Gynec. and Obst.*, 1916, xxii, 127) studied cases while in charge of an American Hospital in France. In the first 225 cases there were thirty-three nerve lesions. Of these, twenty-nine were injuries to the peripheral nerves. The musculospiral was involved 8 times, the median 4, the ulnar 6, the external popliteal 2, the circumflex 1, the musculocutaneous of the leg 1, the sciatic 1, the brachial plexus with sensory disturbances in the ulnar 1 time. Other lesions complicated the nerve injuries in some cases. He says that damage to an important peripheral nerve is an injury of extreme gravity. Primary nerve suture is rarely indicated in war time. Unrelieved, overstretched, muscular tissue leads to fatty degeneration and consequent loss of contractility. A paralytic deformity with shortened muscle and limited joint movement, in the majority of cases, is the result of ignorance or neglect. It is imperative, whether the nerve is divided or not, that the paralyzed muscles be relaxed and protected from strain by a suitable apparatus. Under no circumstances must this be deferred to the so-called after-treatment. The postural prophylaxis begins with the reception of the wound and continues after the operation until voluntary motion is restored. A strict adherence to this vital orthopedic principle aids in the diagnosis, hastens recovery, prevents many distressing deformities, and will materially diminish the number of useless limbs. This essential principle has not received the attention from the general profession that it deserves.

The Operative Treatment of Varicose Veins and Ulcers Based upon a Classification of These Lesions.—HOMANS (*Surg., Gynec. and Obst.*, 1916, xxii, 143) says that varicosity of the veins of the legs is confined for anatomic and physiologic reasons to the superficial and perforating vessels. Trendelenburg's tests distinguish between pure surface varix and surface varix complicated by varicosity of the perforating veins, a distinction for purposes of treatment. Surface varix is curable by relatively simple surgical procedures, preferably excision of the great

saphenous vein from groin to mid-calf. Surface varix complicated by varicosity of the perforating veins requires for its cure not only eradication of the great saphenous vein but a thorough exploration of the lower leg in order to ligate varicose perforating veins. Varix following phlebitis is not uncommon, presents a characteristic appearance, is prone to be complicated by varicosity of the perforating veins, and is usually accompanied by obstinate ulceration soon after its establishment. It is a very general rule if not a law, that the more prominent and tortuous the surface veins the simpler the cure; the less noticeable the surface veins the more malignant and resistant their attendant ulcers, and the more radical the operative procedure required for cure. Varicose ulcers, if of long standing and especially if they are surrounded by an area of thick scar tissue, are best treated by free excision and immediate skin graft in connection with the radical removal of the veins to which they are tributary.

THERAPEUTICS

UNDER THE CHARGE OF

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The Therapeutic Action of Iodin.—JOBLING and PETERSON (*Arch. Int. Med.*, 1915, xv, 286) give experimental and clinical evidence that indicates that the action of iodin in causing absorption of necrotic material is due to the lowering of the antitryptic action of the blood. They believe that the antitrypsin of the blood and tissues is the most important factor in preventing the resolution of necrotic tissues such as are found in infarcts and in the caseous areas in syphilis and tuberculosis. They demonstrated that the antitryptic action of the blood and of tuberculous caseous matter is due to the presence of unsaturated fatty acids. They then studied the antitryptic strength of the serum of patients to whom iodids were being given. They found that when iodids were administered a very considerable reduction in the antitryptic activity of the blood occurred. This decrease in antitrypsin permits the ferments normally present in the necrotic areas to become active and thus hasten autolysis. The authors believe that iodin combines with the unsaturated fatty acids, thus causing a neutralization of the activity of these substances as ferment-inhibiting agents. Jobling and Peterson state that there is a general impression among clinicians that iodids are harmful in tuberculosis, as they promote the spread of the infection by causing a softening of the tubercles and also interfere with connective tissue formation. They explain the well known clinical observation that iodids cause tubercle bacilli to appear in the sputum of patients though they were previously absent, by the fact that softened caseous matter ruptures into the bronchi. The softening

of the caseous tuberculous tissue is due to the autolysis induced by the iodin. Pulmonary hemorrhages may be explained in the same way. Clinical experience has shown that iodin is almost a specific in bringing about the amelioration of symptoms and the disappearance of lesions in syphilis, and yet little is known concerning the means by which these results are obtained. As experimental work and clinical observations have demonstrated that the iodids do not destroy the infecting organism we must assume, according to the authors, that the results obtained are due to the power the iodids possess of causing resolutions of the lesions. Their view is that iodin neutralizes the action of the agents which prevent solution and absorption of necrotic tissue, and at the same time lays bare to the action of the real germicidal agent the infecting organism which previously had been protected by necrotic tissue. With the exposure of the infecting organism such agents as mercury and salvarsan would be much more effective.

Mouth Infection as a Source of Systemic Disease.—Rosenow (*Jour. Amer. Med. Assn.*, 1914, lxiii, 2026) says that abscesses or bony changes found about the roots of teeth in various systemic diseases, especially in cases of chronic arthritis, may or may not be primary, but if found the condition should be corrected because vaccine or other treatment largely fails so long as an active focus of infection exists. In the light of our present knowledge the argument that infections in the mouth are very common in individuals in apparent health, does not minimize their importance. Regarding autogenous vaccines, he says that our ideas need to be somewhat revised in view of the fact that the vaccines administered are usually prepared from streptococci in the focus which may or may not be the causative organism. The instances in which good results are obtained are probably those in which the focus harbored the causative organism. The poor results in some cases of arthritis following the use of autogenous vaccines prepared from the tonsils or other presumable focus, may be due to the fact that the organisms present in the focus at the time, were not the same as those actually infecting the tissues. In this case the vaccine would fail to contain the proper antigen and be ineffectual for therapeutic purposes. Rosenow says that he was able to isolate Streptococcus rheumaticus from the stools of two cases of typical rheumatism; in one from an infected ingrowing toe-nail, and in another case from the wound in the thumb following a crushing injury. He cites these as unusual foci of infection and in a given case the determination of a single focus of infection may not be enough to prove the causative factor of the systemic disease.

The Use of Strychnin and Caffeine as Cardiac Vaso Stimulants in the Acute Infectious Diseases.—Newburgh (*Arch. Int. Med.*, 1915, xv, 458) says that for decades many physicians have relied on strychnin in the treatment of certain grave cardiac symptoms occurring in pneumonia, typhoid fever, and other acute infectious diseases. Within the last twenty-five years some physicians have preferred to use caffeine and other drugs when confronted with these symptoms. His communication discusses briefly the question whether failure of either the heart or vaso-motor apparatus is the chief cause of death in the infectious

diseases. He quotes various observations regarding the effect of acute infectious diseases upon the myocardium and the vasomotor apparatus. He sums up the results of these observations by stating: (1) that since there is no evidence that the vaso-motor apparatus is injured in the acute infectious diseases it is not logical to direct treatment chiefly toward this apparatus and (2) that the hypothesis that the heart may be fatally injured in the acute infections, although far from proved, is still tenable in the present state of our knowledge, and that it is consequently logical to attempt to assist it when it seems to be failing. Newburgh investigated strychnin and caffein with regard to their value as cardiovascular stimulants. He concludes that strychnin sulphate, in medicinal doses, does not increase the output from the heart, slow the pulse, or materially raise the blood-pressure, therefore there is no logical basis for its use as a cardiovascular stimulant. Caffein-sodio-salicylate, in the doses employed, does not raise the blood-pressure or slow the pulse. Newburgh points out that his observations do not permit him to state directly whether strychnin and caffein increase the efficiency of the circulation. The fact that the pulse is not slowed by any given drug does not necessarily mean that the drug may not be improving the circulation in other ways. It is equally true that rises of blood-pressure do not necessarily signify better circulation and that an increased flow of blood may occur in the absence of any marked change in blood-pressure.

Report of Thirty-four Cases of Artificial Pneumothorax.—MALL (*Jour. Am. Med. Assn.*, 1915, lxiv, 897) says that the most favorable cases for lung compression are those in which cavity formation has not occurred but which, under usual methods of treatment, are progressing toward softening. In some advanced cases with large cavities, much relief of distressing symptoms may be obtained by compressing the more advanced lung. Removal of fluid with temporary compression by nitrogen gas or air is the most rapid and effective method of treating large pleuritic effusions. Hemorrhage is usually promptly and permanently relieved by artificial pneumothorax. Compression of one lung does not induce increased activity of the disease on the opposite side.

The Treatment of Typhoid with Non-sensitized Vaccine.—CSERNEL and MARTON (*Wien. klin. Wchnschr.*, 1915, xxviii, 229) report encouraging results in treating typhoid fever by intravenous injections of a non-sensitized polyvalent vaccine. The details of the preparation of the vaccine are given in the article. The vaccine was so prepared that each cubic centimeter contained 15,000,000 bacilli and intravenous injections of from 1 to 2 c.c. of the vaccine were given. The intravenous injections were followed in one to one and a half hours by a chill with a decided rise of temperature which soon fell by crisis, the fall being accompanied by profuse perspiration. They believe that this form of treatment is contra-indicated in patients with severe abdominal complications, such as hemorrhage, perforation, cholecystitis, etc., also in patients with irregular heart action and in the markedly delirious and toxic group.

The Preparation of Diabetic Patients for Operation.—Addis (*Jour. Am. Med. Assn.*, 1915, lxiv, 1130) says that the method of preparing diabetic patients for operation by giving them a sugar and starch-free diet is not only useless but dangerous, because, while it may reduce the hyperglycemia and the amount of sugar in the urine, it greatly increases the chances of diabetic coma. When operation is not immediately necessary, and especially in those cases in which the decision as to whether or not an operation shall be performed rests largely on the question as to how much danger would be run by the patient after the operation because of his diabetic condition, it would be a great advantage to have some objective data to supplement the facts relative to this point, which can be gained by clinical observation. The quantity of sugar in the urine is no aid in this respect for the special danger to life is the failure, not of the sugar, but of the fatty acid metabolism. The coma in which diabetic patients die after operation is, often at least, accompanied by the excretion in the urine of large amounts of unoxidized fatty acids, and there is good reason for believing that the condition is due to poisoning by these acids. The estimation of the degree of impairment of the power of the body to oxidize fatty acids is therefore of prime importance in deciding whether or not operation is advisable in any particular case; but the amount of acetone bodies (fatty acids) excreted does not give a reliable indication of the degree of danger, because, although that amount may be small, the reserve power of the body to deal with these substances may be very slight, so that there may be a sudden failure under the special strain induced by operation, with the result that diabetic coma ensues. What is needed is a functional test of the fatty acid oxidizing power. Addis outlines a method which gives a clinical approximation of the total quantity of acetone bodies excreted under abrupt lowering of the carbohydrate content of the diet compared with a period immediately preceding it of moderate carbohydrate intake. The important point to determine is the rate of increase in the excretion of acetone bodies and not the absolute degree of acidosis. However, the absolute amounts as well as the relative increase must be borne in mind. A large relative increase in a patient who was excreting very little acetone bodies under moderate carbohydrate intake will not have the same significance as a similar increase when the previous elimination was high. The fear, excitement and under-nourishment of the patient which frequently accompany operation, brings about a call for the utilization of the food stored in the body. This food consists of glycogen and fat but the most readily available is glycogen, and there will be no very extensive breaking down of fat into fatty acids until the glycogen stores of the body are largely depleted. Even in cases in which the utilization of sugar is very defective, glycogen will diminish the amount of fat required in such emergencies. One aim of treatment should therefore be to bring about a storage of glycogen in the body before operation. The best means yet devised to this end is the oatmeal treatment introduced by von Noorden. The inability of the kidneys to excrete large amounts of fatty acids is a factor in the production of diabetic coma. To facilitate the excretion of the acetone bodies alkalies should be given, preferably soda bicarbonate. It is best to give the alkali in doses of from 5 to 10 grams in plenty of water before each two- or three-

hour feeding with oatmeal and if necessary one or two doses at night. The quantity of alkali required to make the urine alkaline is an indication of the amount of acetone bodies stored in the body, and this point should be considered in prognosis. Neither success in inducing a storage of glycogen in the body before operation, nor in keeping the urine alkaline is an absolute barrier against diabetic coma. They are only palliative measures. Shock is a large factor in the production of coma and Addis suggests that this factor may be obviated by the application of the principles of anoci association.

Technic of Proper Typhoid Vaccination.—KIRSCHBAUM (*Wien. klin. Wchnschr.*, 1915, xxviii, 208) says that the objections to prophylactic vaccination at the present are the resulting symptoms of total pain, headache, fever and general malaise which last from twelve to thirty-six hours. In some cases these symptoms are so severe that the patient does not present himself for the subsequent injections. This objection holds good particularly in war camps as the soldiers are rendered unfit for duty for a period of several days. The author states that these reactions can be avoided by injecting a smaller initial dose and allowing an interval of two weeks between the first and second injections. The vaccine employed by Kirschbaum was prepared from several strains of typhoid bacilli and contained about 600,000,000 bacilli per cubic centimeter. The initial dose of the vaccine is 0.2 c.c., after a two weeks' interval a second dose of 0.8 to 1.0 c.c. is given and after another week the third dose of 1.0 to 1.5 c.c. is given. No reactions followed and Kirschbaum found the agglutinating power of the blood to be the same as with the methods formerly employed.

Vaccine Therapy of Typhoid Fever.—FEISTMANTIL (*Wien. klin. Wchnschr.*, 1915, xxviii, 230) reports a series of 52 cases of typhoid treated by vaccine prepared from a weak strain of typhoid bacilli, according to the method of Besredka. He found that this vaccine retained its full activity for a period of a few weeks only. When given early in typhoid fever it seemed to reduce the duration of the disease markedly, and also a rise of temperature occurring during convalescence could be reduced by the administration of the proper dose of the vaccine. He found no evidence of this vaccine aborting typhoid as is alleged for the serum of Ischikawa. In 10 of the 52 cases various complications developed. Only one patient died, but sixteen patients are still under treatment.

Vaccine Treatment of Typhoid.—EGGERTH (*Wien. klin. Wchnschr.*, 1915, xxviii, 209) reports a series of 48 cases of typhoid fever in which typhoid vaccine prepared according to the method of Besredka was injected into the brachial vein. It was given in doses of 0.5 to 1 c.c. Of 48 patients, 38 were cured, 8 showed no effect and 2 died. The injections were followed by an almost immediate rise in the temperature, from one to one and a half degrees, which lasted from six to ten hours. At the end of this time the temperature fell by crisis, accompanied in many cases by profuse perspiration. Symptoms of collapse were never noted, and there was a decided improvement in the cerebral symptoms, such as headache, dizziness, etc. The best results were obtained in

those cases which came under treatment during the first two weeks and which showed no complications. Of the two fatal cases death occurred within three hours after the injection, and Eggerth says that the autopsies showed sufficient cause to account for these deaths without attributing it directly to the vaccine.

The Treatment of Intestinal Amebiasis.—Jones (*Jour. Am. Med. Assn.*, 1915, lxiv, 982) says that amebiasis may exist in a mild or even advanced form without dysentery, and dysentery may persist for months or years after amebic ulceration without amebiasis. He is convinced that emetin hydrochlorid, while a valuable amebicide, if used alone will not cure amebic dysentery and that relapses will soon occur. The same statement is equally true of ipecac used alone. After an experience with each of these drugs used alone he has reached the conclusion that emetin given by hypodermic, accompanied or followed by the use of ipecacuanha by mouth, will cure most cases of intestinal amebiasis. He believes that emetin hydrochlorid is amebicidal to those entamebas which are in contact with the blood stream, that is, those deep in the ulceration, but would have little effect on those in the intestinal canal or the edges of the ulcerations. The reverse is true of ipecac, the drug given by mouth (by its emetin content) destroying the entamebas in the intestinal canal or outer margins of the ulcerations but not penetrating deeply into the irregular excavations. Ipecac has the disadvantage of producing nausea and vomiting and it has been Jones' experience that emetin hydrochlorid after several administrations produces a tolerance to ipecac which markedly lessens or prevents the disagreeable effects of ipecacuanha. He advises giving emetin hydrochlorid in doses of 0.008 gram by hypodermic for ten days (twice a day for four days and once a day for six days). Ipecac can be started about the eighth day with from 1.5 to 2 gram doses given at bedtime, continued for three consecutive nights and thereafter decreased by 0.3 gram each consecutive night. The disagreeable effects of the ipecacuanha were never manifested. It is necessary to precede the administration of ipecacuanha by tinctura opii in from 0.6 to 1 gram doses. The fact should not be overlooked that emetin is an amebicide and has little to do with the healing of ulcerations. Every case of amebiasis should, after this treatment, be considered one of ulcerative colitis and so treated from a dietetic point of view. At the same time every effort should be made to enhance resistance by change of climate, tonics, etc., to obviate the distressing sequelæ characteristic of the disease.

The Treatment of Pernicious Anemia with Salvarsan and Neosalvarsan.—Bramwell (*Brit. Med. Jour.*, 1915, 2827, 406) presents a further report of 21 cases of pernicious anemia treated by intramuscular injections of small doses of salvarsan and neosalvarsan. He compares his former results obtained by oral administration of arsenic and believes that he has obtained more distinct results by the use of salvarsan and neosalvarsan given intramuscularly. Bramwell says that benefit is more quickly obtained and that the duration of the improvement is generally longer and the proportion of apparent cures seems greater than where arsenic is given by mouth. He prefers the intramuscular

method of giving salvarsan in pernicious anemia, because he believes that intramuscular injections produce a sustained and continued action as opposed to a more temporary effect when salvarsan is given intravenously. Bramwell believes that salvarsan is more effective than neosalvarsan, but salvarsan given intramuscularly usually produces some febrile reaction and more or less pain at the site of injection. The intramuscular use of salvarsan and neosalvarsan have little tendency to give rise to symptoms of arsenical poisoning, especially peripheral neuritis, which sometimes develops under the continued use of arsenic by mouth. Bramwell says that his patients have not been observed long enough to make any definite statement in regard to ultimate results, but the immediate beneficial results, however, in some of the cases, were very striking.

PEDIATRICS

UNDER THE CHARGE OF

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Disturbances of Digestion in Infancy.—JOHN LOVETT MORSE (*New York State Jour. of Med.*, 1916, xvi, 54) classifies the diseases of the gastro-enteric tract into nervous disturbances of the digestive tract, disturbances of digestion and infections. The first condition is caused by irritable or exhausted nerve centres causing vomiting and diarrhea. The second condition may be caused by an excess either in one food element or in the bulk of food, or by diseases or extraneous conditions diminishing the digestive powers. Indigestion with fermentation may develop from disturbances of the normal process of digestion or primarily from excessive bacteria introduced into the intestine. There are marked pathological lesions in this condition as there are in infectious diarrhea. In indigestion caused by too much or too rich a food, the amount or strength of the food should be cut down to a point lower than the normal baby of that age would be taking, and then raised carefully to the point of tolerance again when the disturbance has disappeared. The point of tolerance is best determined by examination of the stools. When individual food elements are cut down the other elements must carefully be raised to keep up the caloric value. In the case of sugar another sugar may be substituted. In protein excess regulation of the mother's life, or in the bottle-fed cases, diminishing the casein percentage, boiling the milk, addition of alkalies or citrate of sodium and pancreatization may be employed. Drugs are of little or no use in simple indigestion. Cleaning out the bowel with castor oil and by the use of enemata is an important part of the treatment. Indigestion with fermentation may be caused by the butyric acid bacilli, the colon bacillus, and even the normal lactic acid forming

organisms. It is more serious than simple indigestion, more often acute and accompanied by fever. Vomiting is uncommon while diarrhea is marked. Usually the carbohydrates and less often the fats are acted on, causing the fermentation. Lactic acid bacilli are indicated in treatment where the carbohydrates are selected by the attacking organisms, and may be given in broth cultures or in modified milk ripened by them. They are also indicted in the proteolytic type of condition. In the carbohydrate type this element should be cut down and *vice versa*. Symptoms indicating a possible fatal outcome are excessive vomiting, hyperpyrexia, irritation of the central nervous system and collapse.

Influences of Syphilis on the Chances of Progeny.—M. Bishop Horman (*British Med. Jour.*, 1916, No. 2875, p. 196) in investigating the causes of blindness in 1100 children also collected the family history in 150 families. In all of these families one or more children were undoubted sufferers from congenital or inherited syphilis. Besides the state of blindness there were other indubitable signs of syphilis in Hutchinsonian teeth, characteristic physiognomy, scars, bone and joint disease, deafness, etc. Whatever the evidential value of blood tests for syphilis there is certainty in this evidence presented by the finding of these other clinical features. Hutchinsonian teeth alone give certainty and association of this form of teeth with one or more of these other clinical characters provides a superabundancy of certainty. In the 150 families above referred to, 68.5 per cent. Hutchinsonian teeth were present in the blind child of the family. Where evidence of the teeth was lacking there were the other symptoms in sufficient number to explain the blindness. Compared to the 150 syphilitic families a second series of 150 families were studied in which the women were not syphilitics or the progenitors of syphilitic children. The results of fertilization of these two series of women show that 150 syphilitic mothers had 1001 pregnancies, but of these only 390 resulted in healthy children, while the 150 healthy mothers had 826 pregnancies which resulted in 654 healthy children. There were 92 miscarriages per 1000 among the syphilitic series against 73 among the healthy, and 80 stillbirths per 1000 among the syphilitic against 20.5 per cent. among the healthy women. These figures speak sufficiently of the influence of parental syphilis on the chances of healthy progeny.

A New Treatment for Pertussis.—Rudolf Kraus (*Wien. klin. Wchnschr.*, 1915, xxviii, 1405) has attempted a new principle in the therapy of infectious diseases which rests on the idea that the reactions from bacterins, etc., are caused, not by the bacteria themselves but by albuminoid bodies. He has already shown that a watery extract from agar cultures of bacteria coli gives the same results in puerperal infection or typhus as does the bacterium itself. The application of the new idea was made on pertussis. The Bordet-Gengou bacillus has been claimed by many observers to be the true cause of pertussis and been used for serum and bacterin therapy in the treatment of the disease. The results have not always been encouraging and with the idea of the efficacy of albuminoid bodies in mind Kraus utilized the sputum of children with pertussis for therapeutic purposes. The sputum of the

early paroxysmal stage was collected in sterile dishes, washed, tested for tubercle bacilli, mixed with ether, and shaken for one day to eliminate the possible spirochetes of syphilis. The ether was evaporated at 37° C. and the liquid tested for sterility, aërobic and anaërobic, and by injection into animals. The sterile fluid was then injected subcutaneously, from 0.5 c.c. to 2 c.c. every third or fourth day. No local reaction was observed. The experiments were carried on by different observers in three separate hospitals and 50 children from a few weeks to fourteen years of age, all suffering from typical pertussis in various stages, were treated in this manner. The results showed that almost invariably improvement began after the second injection, the paroxysm rapidly diminished in number and became more catarrhal in character, the vomiting disappeared rapidly in all cases and where hemorrhages were common they rapidly disappeared. In general the result was a marked improvement. The chronic cases of two or three months' standing were equally improved by this treatment as were those cases complicated by bronchopneumonia and measles. No other form of treatment previously used showed such a degree or rapidity of improvement.

OBSTETRICS

UNDER THE CHARGE OF

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The Prevention and Treatment of Perforation of the Uterus Complicating Abortion.—SCHWEITZER (*Monatschr. f. Geburtsh. u. Gynäk.*, 1914. xlii) reports from Zweiffel's Clinic in Leipzig 8 cases of perforation of the uterus complicating abortion. He has collected 105 in the literature with a mortality of 25 per cent. Where such patients received no treatment, the mortality rises to 50 per cent. The question as to whether the uterus in abortion is especially vulnerable has occasioned much discussion. It has been abundantly proved that there is a change in the uterine muscle in early pregnancy, so that an instrument can be inserted more readily than normal, and it is often observed that stitches do not hold and that tenaculum forceps tear out. A series of cases has been reported where pathological conditions of the uterine muscle have been found after perforation of the uterus complicating abortion. Thus a blighted ovum or molar tumor weakens the wall of the uterus through the overgrowth and penetration of the villi of the chorion. When one comes to consider how this accident can be avoided, solution of the problem is not so simple. Free manipulation upon the pregnant or puerperal uterus should be made as gently and carefully as possible. In all cases where the uterus is strongly bent, perforation may readily occur during curetting or packings. Where this accident happens in hospitals, and in the hands of good operators, it is very seldom followed by bad results. The important point seems

not to be the instrument used, but whether or no the instrument employed has been correctly used. The finger of the obstetrician may produce this lesion. So laminaria tents when introduced or when withdrawn may perforate the uterus. The effort to pack the uterus after removing the tent, which has weakened the wall of the organ, may result in rupturing the uterine wall and infecting the surrounding tissue. Where a tent disappears into the uterine cavity, it would be better to incise the uterine wall to remove it than to run the risk of injury by unskilful manipulation. So Hagar's dilators, although ordinarily a safe instrument, have repeatedly caused laceration and perforation. Very often the point of perforation is just above the internal os, so that the greater part of the uterine cavity is not in communication with the cavity of the pelvis. Cases where Hagar's dilator causes the accident seem to be especially prone to the development of infection. The most dangerous instruments, however, are those used to remove portions of the ovum. Often the fact that the cervix is not properly dilated predisposes to this accident. In endeavoring to insert the placental forceps in a narrow cervix, the tissues suddenly yield and the instrument is forced through the wall of the uterus. Especially is this liable to happen where there is a sharp flexion. Small, sharp curettes are especially dangerous. It seems possible at times to remove the small ovum with a curette, but at four or five months this cannot be done. The effort to do so will inevitably result in injury. Even in the early months of gestation the curette cannot be relied upon to empty the uterus, and the womb in these cases should always be explored with the finger. The effort to do curetting without exposing the patient and without placing her on a suitable bed or table, cannot be to strongly condemned. As complications, perforations of the intestine or wound of the intestine are especially dangerous. This, in 48 such cases, 13 died (a mortality of 27 per cent.). The injury was 25 times in the duodenum, 16 in the sigmoid flexure, 6 in the ascending and transverse colon, and in 2 in the appendix. Simple prolapse in the intestine through the rent in the uterus is not necessarily of great importance, provided it is promptly detected and the intestine replaced. Prolapse of the omentum is not of especial importance. In 2 cases the tube and ovary were injured; in 1 the urinary bladder; in 1 the right ureter and pelvis of the kidney, so that the kidney had to be removed. The patient fortunately recovered. In 1 case the promontory of the sacrum was injured by violent and unskilful manipulation. When there is a history of interference and manipulation within the uterus in a case of abortion, the case must be considered as at least a suspect from the standpoint of infection. In preventing this accident, one must take especial care to thoroughly dilate the cervix before attempting any intrauterine manipulation. A thorough examination under ether before proceeding to dilatation is indicated. In the first pregnancy the writer prefers the laminaria tent, replaced and gradually increased in size until the finger can explore the entire cavity of the uterus. A packing of iodoform gauze, to remain not longer than twelve hours, is indicated. After the fourth month he would employ Tarnier's dilating bag, although prior to that time he would carefully avoid the use of the bag. To empty the uterus he would employ the finger only. If perforation is diagnosticated, further manipulation must immediately

cease, though douching of the uterus is permissable, and some cases can be successfully treated by an ice-bag, the giving of opium and an intra-uterine pack of iodoform gauze. In 17 cases of perforation of the uterus complicating abortion, 9 died; 6 of peritonitis. One died of general sepsis, 1 of ileus, 1 of pelvic abscess, 1 had peritonitis but recovered, 1 had absscess in the pelvis, which became encapsulated and three weeks later burst into the peritoneal cavity. In 4 cases there developed a fistula between the uterus and the intestine. Of these 4, 1 died of cachexia and 3 recovered. One patient died in collapse. But 2 cases in 17 treated expectantly without operation recovered without serious complication. The mortality of cases of perforation of the uterus complicating abortion, treated without operation, is 53 per cent. These results indicate that operation should be the rule. Section should be done, and Ringer's solution should be employed to cleanse the peritoneum. If the uterus is but little injured and there is no evidence of infection, the wound in the uterus may be closed by suturing and the abdomen closed with a pelvic drain for a short time. The effort to make the operation extraperitoneal, has frequently resulted in the formation of abscess in the abdominal wall. In cases where infection is feared, radical treatment naturally consists in the removal of the uterus. In 45 cases treated by section and extirpation of the uterus, there were 9 deaths, 6 of which were from peritonitis. Of the remainder, 1 was from collapse, 1 hemorrhage, and 1 from an unknown cause. The deaths from peritonitis in those treated conservatively showed the mortality-rate of 11 per cent. while of those treated radically by operation, the mortality for peritonitis was but 2.8 per cent. There is danger in performing a superficial amputation and dropping the stump, and in 8 of these cases, 2 died of peritonitis. These were treated by vaginal extirpation of the uterus with one death from peritonitis. The presence of injury to the intestine increases the mortality 7 per cent. Of 24 treated by total abdominal extirpation, there was one death by peritonitis—4.2 per cent. The evidence seems to point clearly to the value of operative treatment, and that in the majority of cases this must be radical and not conservative.

The Abderdalden Test.—As experience increases with the Abderhalden test, it becomes evident that it cannot be considered as proof positive of pregnancy, but that numerous other conditions may produce it. ABELER and LÖHNBERG (*Berl. klin. Wchnschr.*, 1915, No. 13), have tested this method with the extracts made from carcinomatous tissue and from connective tissue of the kidneys. Their series of experiments numbered 160. In none of them did they use for normal serum material obtained from the placenta. In 11 cases of extra-uterine pregnancy, 6 gave a positive, 5 a negative reaction to ninhydrin. With other observers they found that in some cases a simple hematoma of the pelvis would give a positive reaction, while in others an inflammatory tumor of the adnexa would give a positive reaction to placental extracts. In 39 cases of normal pregnancy, the method was positive in but 1 and this patient had some grave disturbance in metabolism. Among patients who were not pregnant but suffering from some pelvic disease, positive results were obtained in 19.6 per cent. Patients who had fever or tumors gave a positive reaction to placental tissue. In

the carcinoma material 82 per cent. gave a positive reaction. In general the observers believe that the Abderhalden test gives an equally good result in both pregnancy and carcinomatous disease, but to distinguish between these, has not yet been clearly determined. KEITLER and LINDER (*Wien. klin. Wchnschr.*, 1915, No. 21) in 24 cases examined, in 19 used serum obtained from placental substance only. One case gave a negative result where a carcinoma was present and others gave positive results when nitrogenous material, placental or carcinomatous, was present. In 21 cases of pregnancy the case behaved in a characteristic manner in 11. In 27 cases of carcinoma the result was clear in 15. In 21 cases of myomatous tumor, the specific reaction was obtained in 11. In these, as a substratum, the middle muscular layer of myofibromatous tumor was employed. In a series of observations made, the results of the test were far from ideal. Frequently the fault lay in some failure to carry out completely the necessary technic.

Criminal Abortions.—BENTHIN (*Ztschr. f. Geburtsh. u. Gynäk.*, 1915, lxxvii, 3) has studied the frequency and results of criminal abortion from the material in Vinter's Clinic in Königsberg. He finds that at least from 19 to 22 per cent. of all abortions are criminal. All classes of society are embraced among these and the mortality from septic infection far exceeds that of ordinary parturition. The morbidity in these cases is very great and the patient's general health and ability to bear children subsequently and her power for work are very greatly damaged. These cases seem to occur about equally among the married and unmarried. Regarding the means of preventing these occurrences, Benthin warns against the prescription by physicians of vaginal douches taken by patients at their houses and also against the use of intra-uterine pessaries. Indirectly, whatever can be done to regulate and improve the guild of midwives and stamp out the work of those unreliable and disposed to do criminal operations, will also help greatly. Private maternity hospitals should be carefully supervised and all objectionable establishments should be permanently closed. All cases of abortion followed by fever should be immediately reported to the health authorities.

The Mortality from Puerperal Sepsis Since the Year 1900.—BUESS *Ztschr. f. Geburtsh. u. Gynäk.*, 1915, lxxvii, 3) gives the results of his investigations on this subject from von Herff's Clinic at Basel. He concludes an extensive study of this subject by giving the statistics from twenty cities and countries, including Sweden, Italy, Germany, Norway, England, Switzerland, Austria, Paris, Berlin. The lowest mortality rate is that of the city of Basel, which he states at 0.47 per cent.; the highest that of Berlin, 5.44 per cent. England has 1.51 per cent.; Paris, 4.30 per cent.; Madrid, 3.42 per cent.; Vienna, 3.28. In recent years there has been a diminution varying from 0.01 per cent. to 0.87 per cent. in various cities and countries quoted. von Herff adds to the preceding the mortality in puerperal sepsis in Basel Clinic in 16,999 women from 1902 to 1913. In 84.76 per cent. there was absolutely no fever during nor after parturition. In 15.24 per cent. the temperature rose above 101°. The mortality was 0.58 per cent.

GYNECOLOGY

UNDER THE CHARGE OF

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Practical Points in the Technic of Pyelography.—A few minor, but exceedingly practical points for eliminating as far as possible all dangers from the performance of pyelography have recently been brought out in a short paper by WOODRUFF (*Surg., Gyn., and Obst.*, 1916, xxii, 241). The author's main plea is for *care* and *gentleness* throughout. He emphasizes very strongly that the entire cystoscopy should be carried out *directly under the x-ray tube;* "this," he says, "is the most important point in the whole procedure, and to disregard this point causes great discomfort to the patient and possibly death. The best arrangement is to have the cystoscopic room next to the *x*-ray room, so that wires and a window may be cut through and a tube used at any time in the cystoscopic room. . . Where this arrangement is not feasible the *x*-ray room must be used." Woodruff believes that most of the injuries and deaths reported have been due to careless injecting and subsequent rough handling of the patient, and that after the collargol or other fluid has been injected the patient should be kept absolutely quiet until all manipulation has been done and the fluid has drained away. The bladder is emptied of all cystoscopic fluid in order to remove any pressure, and the patient is permitted to lie quietly on the table for half an hour after the picture has been taken to allow the kidney pelvis to empty itself. It is particularly to be insisted on that no pain shall be caused by the injection, as this implies overdistention of the pelvis. The fluid is allowed to run in entirely by gravity, the container being held in the hand of the operator so that it can be instantly raised or lowered as may be necessary. As soon as the fluid is seen trickling from the ureter alongside the catheter the picture should be taken and the injection stopped. In working with thorium solution, instead of collargol, Woodruff adds a small amount of methylene blue, so as to be able to detect it when it begins to flow from the ureteral orifice, as the thorium itself is colorless. (The use of thorium instead of silver salts, such as collargol, for pyelography, as suggested by Burns, was discussed in this department a few months ago.)

Fatal Outcome of an Attempt to Form an Artificial Vagina.—Of recent years numerous reports have appeared in the literature of operative procedures for the formation of an artificial vagina in cases where this structure is undeveloped or absent. In practically all these cases, the purpose has been merely to make possible the act of coitus, the internal organs in such patients being always of such rudimentary type as to preclude the possibility of impregnation. Practically all the methods that have been accompanied by any degree of success depend upon the utilization of some portion of the intestinal canal to

form the walls of the new vagina, since nearly if not all attempts to form a canal lined merely by skin flaps have resulted in such extensive contraction that in a short time the canal has failed to serve the purpose for which it was created. It follows, therefore, that operations for the formation of an artificial vagina involve of necessity opening the peritoneal cavity and interrupting the continuity at some point of the intestinal tract, a procedure that must of necessity be accompanied by a very decided percentage of risk to the patient. It is rather to be wondered at, then, that numbers of reports of serious or even fatal consequences of such operations are not on record; a recent report from the University Gynecological Clinic of Berne, by GUGGISBURG (*Zentralbl. f. Gynäk.*, 1915, xxxix, 827), illustrates, however, one of the serious dangers accompanying these operations. The patient, aged twenty-eight years, and engaged, was anxious to be married. There was no trace of a vagina, notwithstanding which fact, the patient had contracted a severe urethral gonorrhea. After this was completely healed, an operation of the typical Baldwin type was undertaken for the purpose of establishing an artificial canal. After a fossa about 10 cm. deep had been established by blunt dissection between bladder and rectum, the abdomen was opened, and a loop of intestine about 35 cm. long isolated from the ileum, continuity of the intestine being established by lateral anastomosis. This anastomosis was about 20 cm. from the ileocecal valve. Both ends of the isolated loop were closed, and the middle of the loop drawn down into the tract previously formed between bladder and rectum. The wall of the intestinal loop was then stitched to the vulvar orifice, and the intestine opened. The patient did well for three days, but then began to show signs of peritonitis, and died on the sixth day. Autopsy showed a diffuse peritonitis with complete gangrene of the isolated segment of intestine, evidently the result of constriction of the mesentery at the point where it passed through the peritoneum of Douglas's pouch.

Autotransplantation of the Corpus Luteum.—The question of ovarian transplantation has occupied of recent years a position of considerable interest among gynecologists, and has been discussed at various times in these pages. In most of the reported cases, the attempt has been made to retransplant into some portion of the body ovarian tissue obtained from the patient herself in cases where it was not thought advisable to leave either ovary *in situ*, because of inflammation or other pathological conditions, the purpose of this being to prevent the nervous phenomena which often accompany the artificial menopause. A report of two experiments along a somewhat different line have recently been reported by DE LEE (*Surg., Gyn. and Obst.*, 1916, xxii, 80); while both resulted in failure their distinctly novel character makes them worthy of passing notice. In both instances the patients were women in the second month of pregnancy, who were operated upon for large unilateral ovarian cysts, the corpus luteum of the pregnancy being located in the cystic ovary in such a position that it was impossible to avoid removing it. In view of the well-known theory of Fränkel and others that the continuation of pregnancy, at least in the early weeks, is dependent upon the persistence of the corpus luteum, De Lee made an exceedingly ingenious attempt in both cases to preserve the

pregnancy by excising the corpus luteum from the extirpated cystic ovary and reimplanting it between the layers of the broad ligament. One patient went for four weeks following operation, and then had an abortion accompanied by sudden relaxation of the uterus and very alarming hemorrhage, from which she eventually recovered; the other patient aborted on the twelfth day, accompanied also by rather severe hemorrhage. It would seem probable, as far as one can judge from these two cases, that the transplanted corpus luteum had been rapidly absorbed, and had been of no functional benefit to the patient.

Unusual Case of Vicarious Menstruation.—A remarkable and exceedingly interesting case of vicarious, or as the author prefers to term it, "compensatory" menstruation, is reported by CONDIT (*Amer. Jour. Obst.*, 1916, lxxiii, 238). The patient was subjected to a hysterectomy with complete removal (supposedly) of both tubes and ovaries for extensive pelvic inflammation consequent upon a criminal abortion performed two years previously. Two weeks after the operation (the patient's regular time for menstruation) she had all the menstrual molimena, accompanied by a hemorrhage into a small nevus situated over the left ninth intercostal space. Originally the size of a split pea, in a few days the nevus was as large as a hen's egg, with considerable ecchymosis in the surrounding skin. There was no exudation of blood, however, and the tumor rapidly diminished to half its former size. This phenomenon was repeated regularly for twenty-one months, each hemorrhage causing an additional amount of infiltration, until a tumor as large as a child's head had developed, presenting a strong resemblance to a mammary gland. Eventually the tumor ruptured during one of the menstrual periods, accompanied by an alarming venous hemorrhage. It was excised, and was found to consist of dilated bloodvessels, many of which were ruptured, with extravasation of their contents into the surrounding tissue. At the time of the next following regular period the patient's left breast began swelling until it reached twice its normal size, the skin becoming ecchymotic, and the entire organ presenting a bluish-black appearance. The repeated attacks upon this breast became exceedingly alarming, and were attended with severe pain in the mamma and axilla. The gland always subsided almost to normal between the periods, and there was never any discharge of blood or other secretion from the nipple. After about a year the attacks became less severe, and by the end of two years had ceased altogether so far as the breast was concerned. Some time later, however, the patient was seized with extensive subcutaneous hemorrhages into the extensor surfaces of both legs, accompanied by pain and extreme ecchymosis from the inguinal region to the knees. These attacks occurred at irregular intervals for several months, the last occurring in July, 1914, seven years and three months after the hysterectomy. The author considers this remarkable case due to a struggle on the part of the physiological organism to compensate for the loss of the uterus, the stimulus being provided by secretion from a small portion of an ovary left at operation or from a supernumerary ovary. He considers increased vascular tension, and disintegration of blood elements important factors, and that the various vascular areas were attacked in the order of their least resistance.

HYGIENE AND PUBLIC HEALTH

UNDER THE CHARGE OF

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Rat-bite Fever.—Rat-bite fever is a paroxysmal febrile disease of the relapsing type following the bite of a rat. It is due to the Streptothrix muris ratti, first described by Schottmüller in 1914. The wound heals readily but after a variable incubation period of a few days to a month the wound becomes inflamed and painful. Lymphangitis and adenitis set in and are quickly followed by symptoms of systemic infection ushered in by a chill and rapid rise in temperature. A characteristic exanthem of bluish-red, erythematous, sharply marginated macules appears, generally distributed. About 10 per cent. of the cases terminate fatally, usually during the first febrile period, occasionally later from nephritis or exhaustion. BLAKE (*Jour. Exper. Med.*, 1916, xxiii, 39) reports the isolation of a streptothrix from the blood during life and at autopsy in a case of rat-bite fever. The organism is a true branching, filamentous organism, showing fragmentation of the myocelial filaments, granule formation, and the development of chains of coccus-like forms. It is Gram negative and not acid-fast. It is a facultative anërobe, non-motile, non-chromogenic, without capsule or spore formation. It grows in discrete colonies on Loeffler's blood serum, blood agar, and ascitic agar, and as a flocculent sediment in ascitic bouillon. No growth occurs on other media. It is only slightly pathogenic for rabbits and white rats, producing in some cases a local inflammatory and proliferative reaction at the site of inoculation and general lymph node hyperploria. The organism is identical with a streptothrix isolated from a case of rat-bite fever reported by Schottmüller in 1914 and designated by him Streptothrix muris ratti. The organism was agglutinated by the patient's serum in dilution as high as 1 to 320, a fact which further substantiates its etiological relationship to the infection in this case. Pathological examination showed an acute ulcerative endocarditis of the mitral valve with masses of streptothrices in the vegetations, and infarcts of the spleen and kidney. There were subacute lesions of the myocardium, liver, adrenal, and kidneys all of a similar nature, consisting of areas infiltrated with polynuclear leukocytes, lymphocytes, plasma, and endothelial cells with varying degrees of degeneration of the normal cells of the affected area. From the results obtained in the study of his case, Blake concludes that rat-bite fever is a specific infectious disease caused by Streptothrix muris ratti; that the organism invades the

blood stream; that the host develops a protective mechanism against the streptothrix as shown by the presence of agglutinins; and that pathological changes of an inflammatory nature occur in the heart, kidneys, liver, and adrenals.

The Control of Cholera.—McLaughlin (*Am. Jour. Trop. Dis. and Prevent. Med.*, 1916, iii, 392) points out the modern method for the control of Asiatic cholera on international trade routes. Instead of the ordinary period of detention of five days in quarantine, persons may be released upon a negative stools examination. This method was made official upon the recommendation of Dr. McLaughlin by a government regulation, promulgated July 19, 1911. The importance of carriers in cholera is emphasized and the following method recommended for their detection: Specimens are best obtained by administering a saline or by using a rectal tube with "eyes" cut into it. The specimen is then planted in peptone, incubated at 25° to 30° C. for six hours, and examined not later than eight hours. A smear is made from the surface of the peptone growth, avoiding any pellicle. The smear is dried slowly and fixed with heat, stained with carbol-fuchsin, and diluted 1 to 9. It is then examined for curved or vibrio forms. All positive or suspicious specimens are plated on ordinary nutrient agar (2 to 3 per cent.) neutral to phenolphthalein, the surface of which has been dried by an hour in the thermostat. These plates are examined after about sixteen hours. Suspicious colonies are fished and mixed with specific cholera serum, 1 to 600, which should agglutinate true cholera at once. The colony is also transplant upon an agar slan for further study.

The Trend of American Vitality.—Statistical studies plainly indicate that during the past ten years in the United States there has been a decrease in mortality in age groups under forty, and it is clear that this decrease has largely been due to improvement in infant mortality and control of the communicable diseases, especially typhoid fever and tuberculosis. On the other hand there has been an increase in the mortality above forty years. This increase is due to an increase in the incidence of the so-called degenerative diseases. Thus from 1900 to 1910 cancer increased 30.6 per cent.; diabetes, 60 per cent.; cerebral hemorrhage and apoplexy, 18.8 per cent.; organic diseases of the heart, 39.3 per cent.; diseases of the arteries, 396.2 per cent.; cirrhosis of the liver, 14.3 per cent.; Bright's disease, 18.1 per cent.; making an average increase of 33.8 per cent. Many reasons are given for this shortening of the expectotion of life in America after forty years, but a large factor, as pointed out by Dublin (*Popular Science Monthly*, April, 1915), is due to the change in the "population constitution" resulting from immigration from southern and eastern Europe. Statistical studies show that foreign-born as well as the native-born of foreign parentage at the higher age periods and for both sexes have a mortality largely in excess of the native-born of native parentage. The statistics of the degenerative diseases indicate, furthermore, that the nativity factor plays an important part in determining the death rates from these diseases. Thus, both in the registration states and cities where this subject has been studied, it has been found that the native-born of

native parentage show almost uniformly a lower incidence from Bright's disease, diabetes and cirrhosis of the liver than do the foreign-born and their children. The rates, to be sure, vary considerably with the different nationalities; but, taken as a group, the foreign-born apparently show a lower resistance to the degenerative processes which these diseases imply. It is, therefore, not to be wondered at that the death rate in the large cities and States in the registration area shows an increase in mortality in the higher age groups.

Permanent Sediment Records for Water and Sewage.—WHIPPLE and BUNKER (*Amer. Jour. Public Health*, October, 1915, vol. v, No. 10) report a simple method of obtaining a graphic record of the dirt in a water is afforded by the use of some device for straining a given sample through a disk of cotton batting. The sediment retained on the fibers imparts to them a discoloration in proportion to the dirt in the sample. The disk may be dried and preserved by mounting and keeping away from strong light. Any device for accomplishing the filtration is suitable, but the Wizard Sediment Tester for Milk is recommended for ease and manipulation and the uniformity in appearance of its pressed-out cotton disks. For the sake of uniformity the writers recommend the following quantities for filtration as a tentative standard: Filtered water, 1 gallon; ground water, 1 gallon; "clean" surface water, 1 quart; "dirty" surface water, 1 quart; sewage and effluents, ½ pint. This form of record has the following advantages: (1) It can be made easily and quickly; (2) it can be made by any one, laborer or expert; (3) it is inexpensive; (4) the records are self-explanatory and easily understood; (5) within limits, the records are quantitative and may be used for bases of comparison; (6) the records are permanent and may be mounted for preservation and photographed for reduplication, This form of record is recommended as an important addition to routine water analysis records.

The Destruction of Fly Larvæ in Horse Manure.—The United States Department of Agriculture (*Bull. U. S. Dept. Agriculture*, No. 118, July 14, 1914) obtained the best results by the use of borax (sodium borate) and calcined colemanite (crude calcium borate). Both substances possess a marked larvacidal action and appear to exert no permanent injury on the bacteria. In order to kill fly eggs and maggots, apply 0.62 pound borax or 0.75 pound calcined colemanite to every 10 cu. ft. (8 bushels) of manure immediately on its removal from the barn. Apply the borax particularly around the outer edges of the pile with a flour sieve or any fine sieve and sprinkle two or three gallons of water over the borax treated mass. As the maggots congregate at the outer edges of the pile, most of the borax should be applied there. The treatment should be repeated with each addition of fresh manure. Borax may also be applied to floors and crevices in barns, markets, stables, etc., as well as to street sweepings. The borax does not appear to injure the fertilizing value of the manure. Unsatisfactory results were obtained by the use of kerosene, iron sulphate, potassium cyanid, copper sulphate, lime sulphur mixture, Paris green, sodium fluorite, formaldehyde, and the Isthmian Canal Commission larvicide.

The Wassermann Reaction as a Clinical Test, with Special Reference to its Bearing on Matrimony.—HEINMANN (*Jour. Amer. Med. Assn.*, 1915, lxiv, 18, 1463) calls attention to the fact that a positive Wassermann reaction may be obtained in yaws, leprosy, tuberculosis, cancer, malaria and scarlet fever, but that there could be little trouble in excluding these conditions. On the other hand, it is known that alcohol will render a positive Wassermann negative. Acidosis may excite its transitory appearance in individuals in whom it should be absent. He concludes that: The Wassermann is negative at times in active syphilis, but only under definite and characteristic circumstances, and when this is understood, no confusion should arise; the Wassermann test may be positive in the absence of syphilis, in certain other diseases, and under certain conditions easy to recognize and exclude; with these exceptions the positive Wassermann indicates active lues; clinical and experimental corroboration of this point of view exists, and thus assent to matrimony should be withheld from individuals with a positive Wassermann test.

The History of a Typhoid Carrier.—W. A. SAWYER (*Jour. Amer. Med. Assn.*, 1915, lxiv, 25, 2051) reports a very interesting history of a typhoid carrier, responsible for several outbreaks. The carrier was carefully studied over a long period by officers of the Public Health Service and the sanitary authorities of California. The essential features are summed up as follows: (1) Although frequent examinations of the feces of the typhoid carrier H. O. gave negative results for four months after he had been treated with autogenous typhoid vaccine, he infected three persons when subsequently released from quarantine on parole. The total number of persons infected by this carrier, is 30, including 5 who died. (2) In a further attempt to cure this carrier, the gall-bladder and its duct were removed surgically, but the typhoid bacillus was found in the feces several times after the operation. Examination of the gall-bladder showed that it was normal and that its contents were free from typhoid bacilli. (3) After 41 successive examinations of feces during a period of fourteen months, all with negative results, the typhoid bacillus was isolated from stomach contents containing bile. (4) Certain typhoid carriers are unusually dangerous and must be controlled by quarantine or other adequate supervision.

Intraspinal Administration of Antitoxin in Tetanus.—NICOLL, JR. (*Jour. Amer. Med. Assn.*, 1915, lxiv, 24, 1982) has obtained good results in treating tetanus by the following method: (1) From 3000 to 5000 units into the lumbar region of the spinal canal, preferably under an anesthetic, the volume of fluid injected being brought up to 10 or 15 c.c. by the addition of sterile normal saline, the exact amount being regulated according to the age of the patient and the amount of spinal fluid withdrawn. (2) Ten thousand units intravenously at the same time. (3) Repetition of the intraspinal dose in twenty-four hours. (4) A subcutaneous dose of 10,000 units three or four days later. The well-recognized adjuvants to specific treatment—quiet, subdued light, sedatives, etc.—were, of course, understood as a necessary part of the therapeutic measures.

PATHOLOGY AND BACTERIOLOGY

UNDER THE CHARGE OF

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Malignant Tumors of the Thyroid in the Dog.—A considerable variation in the normal structure of the thyroid may be found in the dog. EWALD (*Ztschr. f. Krebsforsch.*, 1915, xv, 85) collected 75 cases of malignant thyroid disease in dogs to which he has added 5 cases of his own. Of these, 63 were cancers, 6 sarcomata, 7 mixed tumors and 4 were undetermined. In about half of these, metastases were found in the lung, while in only 6 were the neighboring lymph glands involved. Because of the frequency of the blood metastases he points to the greater importance of the vascular system of the thyroid as compared with the lymph channels. The secondary growths of these tumors may or may not contain colloid. This he believes is the result of tumor development from two different kinds of cells of the alveolar lining. One type produces colloid, the other does not. Normal cells passing into the blood stream do not become implanted in other organs. He does not believe that the colloid is the active secretion of the gland. The active secretion passes into the blood stream, while the colloid material, representing a useless product enters the lymph vessels. Whether the colloid and the active secretion are produced by the same types of cells is not determined.

Pyelonephritis.—Three explanations have been offered upon the mode of infection of the kidney from the pelvis: (1) by the migration of bacteria upward into the tubules of the pyramids; (2) through systemic infection of the blood; and (3) by way of the perivascular lymphatics passing between the cortex and medulla. RIBBERT (*Virchows Archiv.*, 1916, ccxx, 294) had an opportunity of examining various grades of pyelonephritis in soldiers with spinal cord lesions, who had developed ascending infections of the urinary system. He claims to have observed cases from the earliest pyelitis to those with marked kidney involvement. From this series he was able to follow the stages of progressive inflammation in the kidney substance. Pyelonephritis begins in an inflammation induced by bacteria or their toxins, in the interstitial tissue of the pyramids. A local leukocytosis in the capillaries and veins at the tips of the pyramids is soon followed by an infiltration in the surround-

ing tissue. The bacteria soon invade the neighboring capillaries and veins in which thrombotic masses develop. The bacterial plugs advancing in these vessels as far as the intermediate zone produce an acute inflammation along their course. In the upper portion of the medulla the bacteria pass from the infected vessels to the neighboring loops of Henle. These then become involved in a suppurative process which extends from the medulla into the cortex. Thus the purulent processes lie in streaks following the descending limb of Henle's loop. Along any portion of this route abscesses may be formed. The distribution of the infection in the kidney is confined to lobules having an infected area in the medulla. The glomeruli are not primarily affected. The author does not believe that a continuous upward infection takes place from the papilla to the cortex by way of the tubules.

Spontaneous Primary Tumors of the Liver in Mice.—The occurrence of tumors in individual organs is not uniformly distributed in different species. The frequent presence of cancer of the stomach and uterus in man finds no comparison in animals. Mice commonly have cancer of the mammary gland and the lungs. Primary cancer of the liver is equally infrequent (about 0.3 per cent.) in mice as it is in man. Sarcoma is still less frequent. On the other hand, adenomata of the liver are not uncommon incidental findings in cirrhosis of the liver. Curiously enough, it has been reported that primary cancer of the liver is not uncommon in horses and dogs, while primary sarcoma has been a not uncommon primary tumor of the liver in rats. It has also been reported that these hepatic sarcomata are associated with parasitic cestodes. Slye, Holmes and Wells (*Jour. Med. Research*, 1915, xxxiii, 171) found but 2 cases of tumor of the liver in mice. This appears remarkable in view of the frequency of spontaneous tumors in these animals. In a series of 10,000 autopsies on mice which had died natural deaths, they found 28 growths in the liver of the nature of adenoma. Since many of these mice were under a year old, they believed that the general incidence is considerably higher than 3 per 1000. Fourteen of the animals were males and 14 females. Cirrhosis of the liver was not present. There was no inflammatory process which might be suggested as predisposing to tumor. Of the 28 adenomas, 3 had characters of malignancy, 1 having multiple metastases in the lungs. Three others showed histological characters suggestive of malignancy, while the remaining 22 were benign. None of these tumors had characters of a bile duct new growth. Jaundice and ascites were absent. The tumors have a tendency to appear as multiple primary masses. Ten of the cases showed more than one tumor. Sarcomata were not found in any of the series.

The Development of Giant Cells in the Tubercle.—There are two main theories concerning the origin of the Langhans giant cell: (1) as arising from fixed tissue cells; (2) having an origin from wandering cells, particularly from macrophages. Furthermore there is still lack

of agreement upon the manner in which the multiple nuclei appear within the giant cells. HERXHEIMER and ROTH (*Ziegler's Beitrage*, 1915, lxi, 1) have again attacked the problem by an examination of human material obtained fresh at operation. They believed that from an analysis of many sections, they are able to definitely state that the epithelioid cell is directly derived from fixed tissue cells. These cells, they claim, belong to the connective-tissue group, but they do not exclude in their discussion the possibility of their endothelial nature. They had previously observed the participation of the Eupffer cells in giant-cell formation. They report a curious stellate structure within the epithelioid cells, which may also be found in the various stages of giant-cell formation. It is suggested that the asteroid bodies recently described by many (Wolbach, Vogel, and others) are the degenerated remains of abnormal centrosomes. By appropriate methods such stellate masses may be observed in many giant cells. Their studies have furthermore led them to conclude that the nuclei of the Langhans cells are developed through amitosis and not by the fusion of neighboring elements.

The Part Played by the Acid of the Gastric Juice in the Pathological Processes of Gastric Ulcer.—BOLTON (*Jour. Path. and Bacteriol.*, 1915, xx, 133) believes that the most important factors in the production of ulcer are hyperacidity and motor insufficiency. Both of these factors are the most common manifestations in gastric ulcer. In the present work, he tested out the effect of each of these on monkeys and some cats. In one series of experiments he obtained a gastrotoxic serum by inoculating gastric cells into a goat. This serum when injected into the stomach wall of monkeys gave rise to hemorrhage and ulceration. Under normal conditions these ulcers healed rapidly. When, however, an artificial hyperchlorhydria was induced the ulcers were delayed in healing. Bolton also continued the experiment of serum injection and hyperchlorhydria with an experimental motor insufficiency Under these conditions the ulcers were larger and showed less evidence of repair. This indolency of the ulcer is ascribed to hyperacidity and food retention. When hyperchlorhydria alone was present, a definite chronic inflammation of the mucous membrane was induced. A greater amount of mucus was present on the lining of the stomach and not uncommonly petechial hemorrhages were found. Sometimes these small hemorrhages were associated with follicular ulcers. The various ulcers that he was able to produce in animals were, for the most part, superficial, but sometimes extended to the muscular coat.

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